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FLOW OF METHYL METHACRYLATES[1]

ARTHUR B. GABEL, D.D.S., M.A., AND E. HOWELL SMITH, D.D.S.

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Much of a controversial nature has appeared in the literature on the physical properties of methyl methacrylate. One of us (E. H. S.) has been working with this material for denture bases for some time. Because of this experience and in order to make it easier to avoid bias he continues the selection, examination and processing of the materials while the other of us (A. B. G.) determines the method of testing and collects and analyzes the data.

This paper deals with the general observations on the behavior of these materials without consideration of their nature or method of processing. In the not far distant future it is hoped to present another paper in which these 2 types of data will be correlated. The purpose of this experiment is to study the deformation of methyl methacrylates used in dentistry under a constant compressive load applied over a relatively long period. During the course of the experiment slight digressions of a qualitative nature were made to throw additional light on phenomena observed in the regular course of the work.

The apparatus (*fig. 1*) used was a Tinius Olsen tensile and crushing strength testing machine with a maximum load of 2000 pounds. The load weight and damping cylinder were removed and a small load of suitable size substituted. The load arm was supplied with a pointer which moves over the recording sheet. Recordings were made by marking on the sheet the location of the pointer at suitable time intervals. The specimens were micrometered to the 10,000th of an inch before insertion and after removal and the variations for the different time intervals calculated.

The specimens were cylindrical and approximately .25 inches long by .20 inches in diameter. Variations in these dimensions were unavoidable because facilities for machining were unavailable. However errors resulting from this condition were largely overcome by regulating the load so as to apply a uniform force per unit area and by calculating the percentage change in length where comparisons were to be made. In the beginning the specimens were prepared by numerous individuals and the variations in behavior were extreme. By a process of elimination of types of specimens and numerous preliminary tests a constant load of approximately 7200 pounds per square inch (about twice the load for testing amalgam) was decided upon. This gave the desired results without injuring the material and allowed all readings to be made in a working day.

[1] Read at the 21st General Meeting of the International Association for Dental Research, Chicago, March 13–14, 1943 (*J. D. Res.*, **22**: 204, 1943). Received for publication April 13, 1943.

Some of the poorer or improperly processed material flowed rapidly and at an almost constant rate until the deformation was easily discernable to the unaided eye. It was noted, however, that the better specimens showed a comparatively rapid deceleration of flow. One of these specimens with half the usual load (to use the same value as for amalgam) showed 2.37% shortening in 10 days. Allowed to remain 6 days more, it showed no change.

It had been noted previously that some of the specimens tended to recover so rapidly after removal that it became necessary to micrometer them at definite short intervals and to extrapolate the value for the time at which they were removed from the machine in order to get the true length. However, only specimens subjected to intermittent loading or to continuous loading for short periods (approximately 5 minutes) recovered completely. The rest showed a lag or

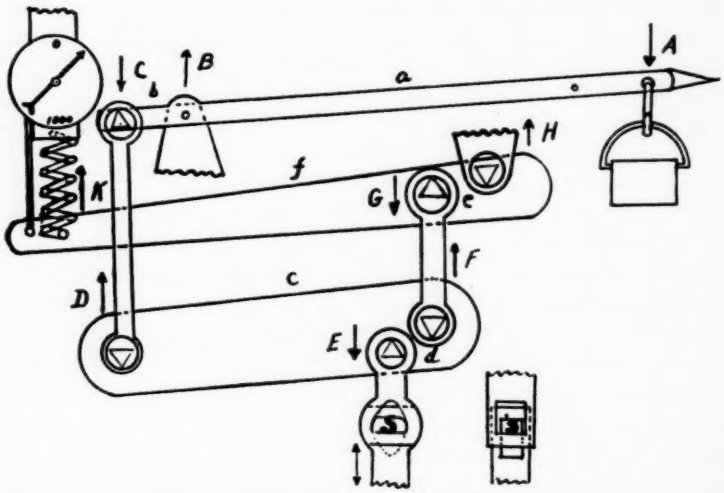

FIG. 1. Diagram of testing machine as modified for recording flow. Constant load applied at *A*, specimen placed at *S*. Distortion registered by movment of pointer at *A*.

hysteresis. One specimen recovered over 25% of its compression in 3 hours and 43% in less than 24 hours.

Several specimens tested by the intermittent application of the standard force used for amalgam, 100 times in about 2 to 3 minutes, showed no distortion or recovered completely in a few minutes. Furthermore the photo-elastic effect observed under similar conditions showed that the strain from some forces of unknown amount disappeared immediately while in some cases it decreased slowly or seemed to remain constant. Because of this tendency to elastic recovery the coefficient of elasticity or restitution against glass was determined by releasing $\frac{3}{8}$ inch spheres of various materials from given heights, allowing them to strike a thick glass slab and observing the height of rebound. The method used was rather crude but the results were consistent and the differences great enough in the case of the acrylic to be significant. The values were about 0.62 for amalgam, coin gold and S.S.W. #5, 0.81 for acrylic and 0.92 for steel.

It seemed probable that a mathematical analysis of the graphs might lead to interesting results. The equations of the graphs obtained in compression and recovery were worked out. The original tests were run for 30 to 45 minutes and the graphs fit a parabolic (*fig. 2*) curve closely. It was noted however that for the higher values of time there was an increasing divergence of calculated and observed values. The tests were therefore run for prolonged periods. The appearance of the graphs for tests that ran practically to the cessation of flow suggested[2] an exponential curve similar to that for an electrical circuit with resistance and inductance, (1), $C = \frac{E}{R}(1 - e^{-\frac{R}{L}t})$. This curve (*figs. 3 and 4*) would not fit the data until an initial distortion was allowed. This initial value corresponded approximately to the values in the parabolic curve where deviation from the formula begins. It would thus seem that there are 2 laws governing

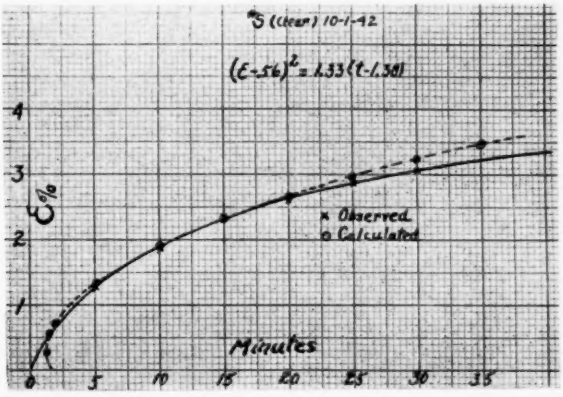

FIG. 2. Typical parabolic curve with formula for early deformation. Note divergence of observed and calculated values after approximately 20 minutes.

the variation of the rate of flow; the first as indicated by the parabolic curve and the second as indicated by the exponential curve. The question as to whether these mathematical entities have a counterpart in physical reality can at present be only a matter of speculation. It must be realized that these curves and their corresponding formulae are empirical.

The apparent fit of the parabolic part of the curve has no physical significance at present and the apparent analogy between the electrical formula or its mechanical analogue should not be carried too far for what we designate as displacement (ϵ) in our equation is current in the electrical case or rate of displacement in the mechanical. The relationship is, therefore, homologous rather than analogous. If the apparent similarity of the second part of the experimental to the electrical curve for which the formula, (1), was given, is real we are dealing with a function whose rate of change is proportional to itself. The

[2] The apparent similarity of the experimental curve to the curve for the electrical case was called to our attention by Dr. T. D. Cope, Professor of Physics in the University of Pennsylvania, who has shown a much appreciated interest in our work.

problem, therefore, is to derive a formula based on this relationship and test the fit of the experimental curves.

In deriving the formula it is more convenient to consider recovery first. It is assumed that the specimen has been subjected to a constant load until flow has ceased. The load has been removed and recovery is about to begin. If the rate of diminution of distortion or rate of recovery, $\left(-\frac{d\epsilon}{dt}\right)$ is proportional at any time (t) to the distortion, (ϵ) then (2) $\frac{1}{B}\left(-\frac{d\epsilon}{dt}\right) = \epsilon$ or (3) $\frac{d\epsilon}{\epsilon} = -Bdt$

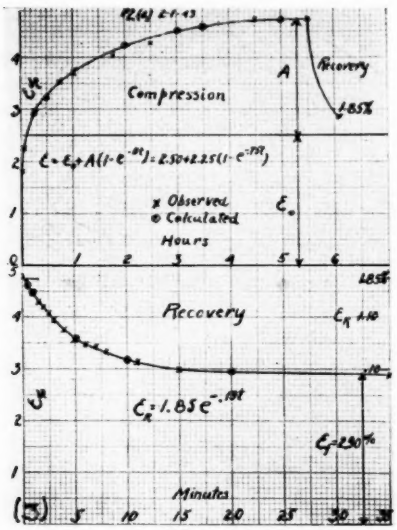

FIG. 3

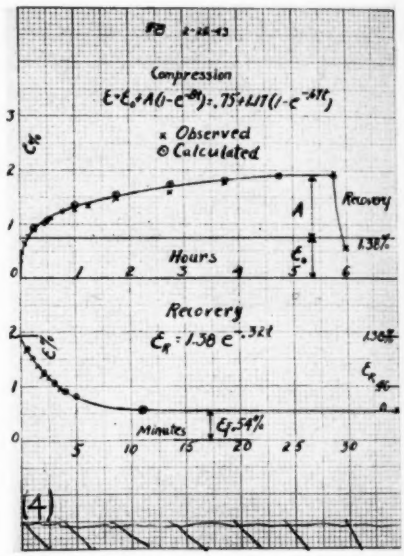

FIG. 4

FIG. 3. Compression and recovery curves with comparatively high compressibility and permanent distortion. Curve at upper right is same as recovery curve below but drawn to same time scale as compression curve.

FIG. 4. Same as for *fig. 3* except that material has comparatively low compressibility and permanent distortion.

where (B) is a proportionality constant which may involve several parameters and which remains to be investigated. Integrating and solving for (ϵ) which we shall now call (ϵ_R) we get (4) $\epsilon_R = Ae^{-Bt}$ where (A) is the total recovery since when $t = 0$, $\epsilon_R = A$ and when $t = \infty$, $\epsilon_R = 0$. It was previously stated that under the above conditions recovery was not complete, a residual distortion (ϵ_f) always remaining. The total distortion at any time (t) is then simply the sum of the unrecovered part of (A) and (ϵ_f) or (5) $\epsilon = \epsilon_f + Ae^{-Bt}$. Equations (4) and (5) fit all the recovery curves for which they were tested.

In compression[3] let the load be (F) and assume that the resistance of the

[3] The method of developing the case for compression follows that for the electrical homologue as developed by John Perry on page 169 in Calculus for Engineers, 12th impression, Edward Arnold, London.

specimen consists of an elastic factor proportional to (ϵ) and a frictional factor proportional to the rate of change of (ϵ). Then (6) $F = k\epsilon + l\frac{d\epsilon}{dt}$. In (5) $\epsilon = \epsilon_f + Ae^{-Bt}$ which is put in the general form (7) $\epsilon = a + be^{-gt}$ as the constants do not have the same value as for recovery since the potential energy of the compressed specimen is less than the work done in compressing it. Furthermore, the (ϵ_f) in (5) is permanent deformation due to flow while (a) in (7) is an elastic deformation. Then (8) $\frac{d\epsilon}{dt} = -gbe^{-gt}$ and substituting in (6) we get (9) $F = k(a + be^{-gt}) - lgbe^{-gt}$ or (10) $F = ka + (kb - lgb)e^{-gt}$. Let $k = lg$ or $g = \frac{k}{l}$ and we have $F = ka$ so that the load (F) may be constant although (ϵ) alters. Substituting in (7) the new values for (a) and (g) we get (11) $\epsilon = \frac{F}{k} + be^{-\frac{k}{l}t}$. Since $\epsilon = 0$ when $t = 0$, $b = -\frac{F}{k}$ hence (12) $\epsilon = \frac{F}{k}(1 - e^{-\frac{k}{l}t})$ which is of the form $\epsilon = A(1 - e^{-Bt})$. This formula fits the second part of the compression curves for which it was tested. For the total distortion $\epsilon = \epsilon_0 + A(1 - e^{-Bt})$. No attempt has yet been made to explain the parabolic portion of the curve. While the physical significance of the parameters implicit in A and B in the recovery and compression formulae are the same respectively the evidence as indicated above is against their having the same numerical value.

SUMMARY

An analysis of our experimental work to date seems to indicate that properly processed methyl methacrylate tends to behave like an elastic material but undergoes a certain amount of permanent distortion under a constant load maintained for an appreciable time. The rate of change of distortion is retarded, apparently by internal friction, so that it has a logarithmic decrement in both compression and recovery. Furthermore, it exhibits an hysteresis-like effect, recovery not being complete. It remains to be determined whether or not this latter phenomenon is comparable to that occurring in the core of an electromagnet.

Many questions remain unanswered. What brings about the seemingly initial distortion represented by the parabolic curve? Is there an instantaneous compression upon application of the load and/or recovery upon its removal which has not been detected because of inherent difficulties? What effects do pearl size and methods of mixing, packing and processing have on the properties being investigated? And so on ad infinitum. Many of these questions must be answered not only to determine whether methyl methacrylate is suitable for certain types of dental restorations but also to obtain the most desirable physical qualities with the materials available.

MECHANISM OF FLUORINE INHIBITION OF CARIES IN THE RAT[1]

RUDOLPH W. NORVOLD, B.S., D.D.S., M.S.D., AND W. D. ARMSTRONG, PH.D., M.D.

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The role of fluorine in promoting resistance of teeth to decay has been supported by evidence from three sources: (a) chemical analyses for fluorine in teeth, (b) studies of the incidence of caries in the human as correlated with the fluorine content of drinking water and milk (1) and (c) animal experimentations. The literature referring to the first two lines of evidence has, for the present purposes, been adequately summarized in other places (2, 3).

Miller (4) reported the rate of appearance of caries in the rat to be decreased by the addition of large amounts of sodium fluoride or sodium iodoacetate to the food. This observation was confirmed by Hodge and Finn (5) who found the daily oral administration of 3 mg. fluorine in solution to rats reduced, not only the incidence of caries, but also the occurrence of fracture of the cusps of the molar teeth. Cox, Matuschak, Dixon, Dodds and Walker (6) reported that the addition of 41.2 p.p.m. fluorine to the diet of rats during pregnancy and lactation brought about an increased resistance of the teeth of the offspring to caries. Cheyne (7) removed the salivary glands of rats and found that fluorine still exerted a protective effect against caries from which observation he concluded that the influence of fluorine is not mediated through the secreted oral fluids. McClure and Arnold (8) observed that water containing 2 p.p.m. fluorine had no effect on the incidence of caries in the rat. However, they noted that 125 p.p.m. fluorine in the form of sodium fluoride in either food or water definitely reduced the caries attack rate in their experimental animals. Arnold and McClure (9, 10) recorded observations which indicated that sodium fluoride administered by subcutaneous injection produced an elevation of the fluorine content of the enamel of the molar teeth of young rats without increasing the caries resistance of the same teeth. They also observed that water containing 10 p.p.m. fluorine furnished only slight, if any, protection against caries in the rat. Sognnaes (11) found a probable reduction of the caries attack rate when concentrated solutions of potassium fluoride were applied to the surface of the molar teeth of rats. McClendon and Foster (12) reported a delay of occurrence of caries in the rat when drinking water containing 20 p.p.m. fluorine was given to rats receiving a diet containing only 0.3 p.p.m. fluorine.

[1] This work was supported by a grant from the Carnegie Corporation while one of us (R. W. N.) held a Carnegie Fellowship in Dentistry. This paper is constructed from a thesis submitted by R. W. N. to the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the Degree of Master of Science in Dentistry.

Received for publication April 13, 1943.

The probable mechanisms by which fluoride reduces the caries attack rate in the human and in the rat have been discussed (2). The available evidence is not sufficient to settle the question as to whether the effect is due (a) to the inhibition of the acid producing bacterial enzymes by fluoride present in the oral fluids, or (b) to the presence of an optimal amount of fluorine in enamel which may reduce its acid solubility or act as an enzyme inhibitor. The possibility has been noted (2) that both of these mechanisms may be involved but the available evidence would indicate, in the case of the human, that the second is the more probable.

A discussion of the mode of action of fluorine in lowering the caries attack rate in experimental animals requires a knowledge of the effect of fluorine supplied to the animals at different times in relation to the development of the teeth and in relation to the regimen on the caries-producing diet. The three important variations of experimental procedure in this regard are: (a) incorporation of extra fluorine in the teeth only during their formation, (b) administration of extra fluorine to rats after the formation of the teeth and a determination of the caries susceptibility while the animals are receiving the extra fluorine, and (c) administration of extra fluorine to rats for a limited time after eruption and maturation of the teeth followed by a determination of the caries susceptibility of the teeth after the fluorine administration has been discontinued. It appears that only two (a and b) of the above mentioned experimental variations have previously been investigated.[2] The third (c) was suggested by the observation of Perry and Armstrong (13) who observed that the fluorine content of the molar enamel but not of the molar dentin was increased when mature rats were given drinking water containing 20 p.p.m. fluorine for 60 days. This observation was interpreted to indicate that the extra fluorine present in the molar enamel entered through the external surface.

The information as to which of the above methods of fluorine administration is effective might serve to indicate the procedure to be used to bring about an increased resistance of the teeth of the human to caries. An additional reason for further investigation in this direction lies in the fact that in the several previous studies the conditions of administration of fluorine, the length of time the animals were fed the caries-producing diet and the methods used in scoring the caries have not been uniform. The time over which the animals were fed the caries-producing diet has varied from 8 weeks (6) to 200 days (5, 7). The methods used for the detection of rat caries in fluorine studies have been a mere examination of the teeth under the binocular microscope (5, 7, 8), a detailed sectioning of the teeth and the employment of a stain (6), and the use of silver nitrate staining of one side of the jaws, this last procedure being checked by grinding and staining suc-

[2] After the manuscript for this paper had been prepared for publication the paper by F. J. McClure appeared (*J. D. Res.* **22:** 37, 1943). McClure demonstrated an increased resistance to rat molar caries produced by fluorine in the drinking water supplied for various periods prior to the regimen on the caries producing diet. The present communication constitutes an extended description of an investigation which had been previously briefly noted in a review by Armstrong (3). The work described in this paper is that noted by McClure on page 38 and as reference no. 18 to his paper.

cessive planes on the other side of the same jaws (4). The result has been that while the conclusions reached by the several investigations noted above are valid no direct comparison can be made between their studies. Thus, even though two of the experimental variations mentioned above (a and b) have previously been investigated it is not possible to determine which of the two is the more effective in reducing the caries attack rate in the rat. In the present investigation all details of procedure have been kept uniform in so far as possible. Furthermore, several methods of examination and detection of rat molar caries have been applied thus permitting a comparison of the significance of the results obtained by these methods.

EXPERIMENTAL PROCEDURES

This investigation attempts to discover the effect of extra fluorine on the susceptibility to caries of rat molar teeth under 3 conditions. (a) In the first case (Group I) the fluorine was supplied to the rats only during the period of tooth formation (during intrauterine life and until 21 days of age). The caries resistance of the teeth of the animals so treated was tested subsequently to the termination of the treatment in order to determine the effect produced by extra fluorine incorporated in the teeth only during their formation. (b) Group II received extra fluorine after the molar teeth were erupted and concurrently with the caries-producing diet. These conditions were similar in this regard to those previously employed by others (4, 5, 6, 7, 8) and were designed to test the effect of fluorine when present simultaneously with the caries-producing factors. (c) Group III was employed to test the effect of fluorine introduced into the enamel of the molar teeth after their eruption.[3] In this case, animals whose teeth were fully formed, were given for 60 days drinking water containing 20 p.p.m. fluorine. This procedure duplicates that of Perry and Armstrong (13) which was shown to result in a significant increase in the fluorine content of the molar enamel of mature rats. After the fluorine administration was discontinued, the caries susceptibility was determined. The possibility that surface application of fluorides to human teeth might confer upon the teeth some degree of immunity to caries has been discussed (13, 14, 15) and lately some attempts have been made to test this hypothesis with humans (16, 17) and with the rat (11). Although the experiments now being reported were carried out in 1940–41, the results with respect to Group III will still have bearing in this connection.

The details of the experimental procedures with respect to the treatment of the animals are summarized in *fig. 1*. It will be noted that one group of animals (Group I_c II_c) served as the controls for experimental Groups I and II. Another group of animals (Group III_c) served as the control for experimental Group III. The diet employed was a modification of that of Hoppert, Webber and Canniff

[3] These experimental studies should be compared with the observational studies of Deatherage (*J. D. Res.*, **22**: 129 and 173, 1943) on the caries rates in individuals living in fluoride water areas during their entire lives, first eight years only, or after eruption of teeth. These papers of deatherage were published after the receipt of this paper. (Ed.)

(18) and was composed as follows: 60 parts yellow corn, 24 parts skim milk powder, 3 parts alfalfa meal, and 1 part sodium chloride. This diet, when coarsely ground was used to produce caries, and when ground so as to pass a 60-mesh screen it was used as the "non-caries" producing diet.

FIG. 1. *Diagrammatic representation of experimental procedure*

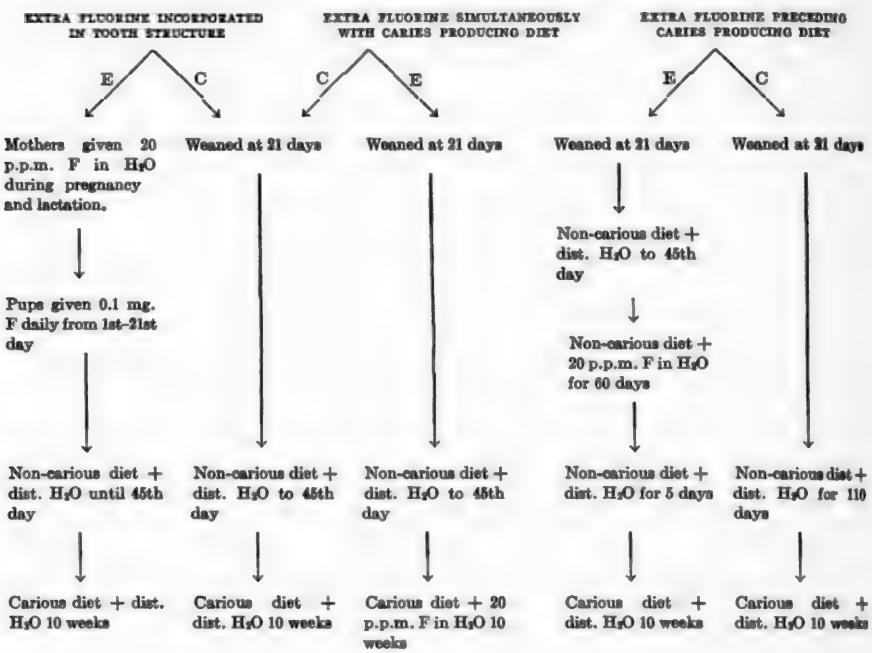
EXTRA FLUORINE INCORPORATED IN TOOTH STRUCTURE
EXTRA FLUORINE SIMULTANEOUSLY WITH CARIES PRODUCING DIET
EXTRA FLUORINE PRECEDING CARIES PRODUCING DIET
E
C
C
E
E
C
Mothers given 20 p.p.m. F in H2O during pregnancy and lactation.
Weaned at 21 days
Weaned at 21 days
Weaned at 21 days
Weaned at 21 days
Non-carious diet + dist. H2O to 45th day
Pups given 0.1 mg. F daily from 1st–21st day
Non-carious diet + 20 p.p.m. F in H2O for 60 days
Non-carious diet + dist. H2O until 45th day
Non-carious diet + dist. H2O to 45th day
Non-carious diet + dist. H2O to 45th day
Non-carious diet + dist. H2O for 5 days
Non-carious diet + dist. H2O for 110 days
Carious diet + dist. H2O 10 weeks
Carious diet + dist. H2O 10 weeks
Carious diet + 20 p.p.m. F in H2O 10 weeks
Carious diet + dist. H2O 10 weeks
Carious diet + dist. H2O 10 weeks
I
Io IIo
II
III
IIIo
26 animals
46 animals
42 animals
40 animals
37 animals

TABLE I

Final weights of animals

SEX	GROUP I	GROUP I_o II_o	GROUP II	GROUP III	GROUP III_o
	gms.	*gms.*	*gms.*	*gms.*	*gms.*
Males	186.0	208.8	196.7	267.5	263.6
Females	139.6	148.8	134.9	180.1	175.1

When the animals were sacrificed the jaws were removed and stored in 50% alcohol. The molar teeth while being observed under the binocular microscope were cleaned of food residue and debris by the use of very fine needles. The average weights of the animals in the several groups at the time of sacrifice are shown in Table I.

EXAMINATION OF MOLAR TEETH AND RECORDING OF OBSERVATIONS

The molar teeth were examined both before and after being stained. The observation method followed in principle that used by Cox, Dodds, Dixon and Matuschak (19) who recognized and numbered 34 caries-susceptible areas in the molar dentition of the rat. The lesions were scored upon a chart which provided for recording the following observations: 1. The total number of unstained lesions visible under 15 × binocular magnification. 2. The number of areas involved by extensive cavitation. 3. The total number of stained lesions visible under 15 × binocular magnification. 4. The number of major caries susceptible areas (exclusive of fracture areas) affected by decalcification as evidenced by staining. 5. Miscellaneous observations.

The staining method used was that described by Gomori (20). This procedure results in (a) a superficial black staining of the bone and the normally exposed dentin at the summits of the cusps, (b) a brown staining of the enamel and (c) a deep black staining of lesions which may be presumed to be caries. Apparently the localization of the deep black stain depends upon fixation of silver by the caries-affected enamel and dentin. Because of difficulty experienced in detecting the blackened areas due to caries in the very small minor fissures, we considered the 20 major fissures only when caries were scored by this method. In the case of areas which were questionably carious, the jaws were ground with a dental engine and stone until a cross-section of the region in question was obtained. The area was then examined under the binocular microscope and a decision made, based upon the presence of the stain in the dentin, as to whether or not the area was to be scored as carious.

DISCUSSION OF RESULTS

In Table II are given the data regarding the total number of molar lesions that could be observed under 15 × magnification with a binocular microscope before staining. In the examination by this method, all of the 34 areas (Cox, Dodds, Dixon and Matuschak) were inspected. It was observed that every lesion that could be seen was apparently either a fracture or was closely associated with a broken cusp in that area. Therefore, since each cusp fracture was recorded on the examination chart the total number of cusp fractures is equal to the number of lesions that were visible. In this method of scoring, the extent to which each individual area was involved was not taken into consideration.

The data in Table II indicate that when the molars of the upper jaws of Groups I and I_c II_c are considered, there is no significant difference between the groups as regards the total number of lesions visible before staining. However, when the lower molars of these two groups are considered, those of Group I show a statistically significant greater number of lesions than those of Group I_c II_c. There can be little doubt but that the lesions of Table I are primarily of the fracture type. Therefore, it would seem that the molars were made more brittle and consequently more susceptible to being fractured in that group of rats which were given fluorine while the teeth were forming. This increased brittleness of teeth containing extra amounts of fluorine has been a common observation in

severely mottled human teeth and has been reported by Armstrong (21) with regard to rat incisors. It is possible that the amount of fluorine administered to the animals was too great and that a lesser amount would not have deleteriously affected their physical properties. Another possible effect noted of fluoride feeding to young rats was that in 11 of the 26 animals of Group I the cusp on the mesio-buccal portion of the upper third molars failed to form. This phenomenon was not observed in any of the rats of the other groups. That the total number of

TABLE II

Total number of lesions observed under 15 × magnification before staining

GROUP NUMBER	NUMBER OF ANIMALS	JAW	NUMBER OF LESIONS PER RAT	STANDARD DEVIATION (S_x)	RELATIVE DEVIATE (k)	PROBABILITY*
Comparison of Groups I and I_c II_c						
						per cent
I	26	Upper	2.42	1.74	+0.56	57.5
I_c II_c	46	Upper	2.20	1.24		
I	26	Lower	10.85	2.92	+3.16	0.16
I_c II_c	46	Lower	8.70	2.62		
Comparison of Groups II and I_c II_c						
II	42	Upper	2.31	1.22	+0.44	65.9
I_c II_c	46	Upper	2.20	1.24		
II	42	Lower	9.00	2.80	+0.53	59.6
I_c II_c	46	Lower	8.70	2.62		
Comparison of Groups III and III_c						
III	40	Upper	2.37	1.40	+2.37	1.78
III_c	37	Upper	1.73	1.03		
III	40	Lower	7.55	2.31	−1.37	17.0
III_c	37	Lower	8.32	2.57		

* Probability of difference of means arising from sampling errors.

fracture lesions in the teeth of the upper and lower jaws of the rats differ is demonstrated very clearly by the observations made upon Group I.

Comparisons of Groups II and I_c II_c with regard to the total number of lesions visible before staining reveals that there is no difference between the two groups other than that which could arise from sampling errors. This seems to indicate that 20 p.p.m. of fluorine administered during the feeding of the caries-producing diet apparently brought about no change in the number of fracture-type lesions.

A comparison of the teeth of the upper jaws of Group III with those of Group III_c with regard to the total number of lesions visible before staining reveals that there were more lesions visible in the teeth of the upper jaws of Group III than in

Group III_c. However, in the lower jaws this relationship was reversed. Considering that in the case of neither the upper nor the lower jaws were the differences between the two groups of statistical significance, one might conclude that the fluorine given for 60 days and then discontinued had no very marked effect upon the subsequent production of fracture lesions.

In Table III are given the data regarding the number of areas of extensive cavitation observed under 15 × magnification before staining. By extensive

TABLE III

Areas of extensive cavitation observed under 15 × magnification before staining

GROUP NUMBER	NUMBER OF ANIMALS	JAW	NUMBER OF LESIONS PER RAT	STANDARD DEVIATION (S_x)	RELATIVE DEVIATE (k)	PROBABILITY*
						per cent
Comparison of Groups I and I_c II_c						
I	26	Upper	0.27	0.44	+.7	48.3
I_c II_c	46	Upper	0.20	0.40		
I	26	Lower	3.42	2.49	+1.48	13.9
I_c II_c	46	Lower	2.50	2.53		
Comparison of Groups II and I_c II_c						
II	42	Upper	0.29	0.54	+.9	36.8
I_c II_c	46	Upper	0.20	0.40		
II	42	Lower	2.50	2.98	.0	100.0
I_c II_c	46	Lower	2.50	2.53		
Comparison of Groups III and III_c						
III	40	Upper	0.30	0.64	+2.7	0.69
III_c	37	Upper	0.03	0.17		
III	40	Lower	0.40	1.18	−1.68	9.3
III_c	37	Lower	0.97	1.91		

* Probability of difference of means arising from sampling errors.

cavitation of an area is meant a gross destruction of one or more tooth areas, regardless of cause and usually involving a pulp exposure.

A comparison of the teeth of the upper jaws of Group I with those of Group I_c II_c indicates that there is no valid statistical difference between these groups with regard to the number of areas of extensive cavitation observed. However, a comparison of the lower jaws of these two groups shows a slightly greater number of these lesions in Group I. This would seem to indicate that those animals receiving the amount of fluorine which was administered while their teeth were forming were slightly more susceptible to the formation of areas of extensive cavitation. As it appears that the formation of these lesions is at least initiated

by the fracturing of the teeth, the number of these areas visible should, and does, roughly parallel the total number of lesions visible in unstained teeth. In much of the investigative work that has been done on experimental rat caries, these areas of extensive cavitation have been designated as caries. It would seem that application of findings of this nature to the problem of human caries would not be valid for the reason that has been mentioned, i.e., that fractures of the molars play a major part in the initiation of such lesions in the rat.

A comparison of the teeth of Group II with those of Group I_c II_c indicates that there is no valid statistical difference with regard to the number of areas of extensive cavitation observed. This indicates that the fluorine which was given simultaneously with the caries-producing diet had no effect upon the production of this type of lesion.

A comparison of the teeth of Group III with those of Group III_c indicates that there is no valid statistical difference between the two with regard to the number of areas of extensive cavitation. It will be noted that the upper teeth of Group III apparently were adversely affected by the fluorine. However, since the lower teeth of these same groups displayed a difference in the other direction and because the number of extensive lesions in both groups was small, it can be concluded that the administration of 20 p.p.m. of fluorine to the animals for 60 days and then discontinued had no clearly demonstrable effect on the incidence of lesions of this type.

In Table IV are given the data regarding the number of stained lesions observed under 15 × magnification. When the molars were stained, it was noted that the effects of the staining were best observed in the major crevices only of the occlusal surfaces of the molars. As indicated above, these major fissures constituted only 20 of the entire 34 areas. In these 20 areas, food particles had the opportunity to become impacted and the acids which have a decalcifying effect upon the tooth structure had the opportunity to form. The action of these acids upon the enamel and dentin was such as to cause these tissues to become black in the staining procedure. In many instances, however, because of the presence of fracture lesions extensive enough to involve completely one or more of these major fissures, it was impossible to determine whether the staining of the adjacent tooth structure was brought about by the effect of acids and is thus a true carious process or by the deterioration of the tooth tissue resulting from extensive fracture and subsequent destruction of the adjoining tooth tissue. Areas involved in the last manner were recorded such that they could be segregated from the stained areas which were not affected by fractures.

As Table IV includes all of the stained lesions involving the major fissures, it undoubtedly includes not only the occlusal fissure lesions which perhaps are comparable to human caries but also the lesions which were initiated by fractures. The data given in this table should thus be considered accordingly.

A comparison of the molars of Group I with those of Group I_c II_c in Table IV indicates that there is no valid statistical difference between these two groups with regard to the total number of stained lesions observed. Thus the fluorine given to the rats while their teeth were forming apparently had no effect upon the

subsequent production of the total number of stained lesions. A comparison of the teeth of Group II with those of Group I_c II_c indicates that 20 p.p.m. of fluorine given to the animals simultaneously with the caries-producing diet apparently lowers the production of lesions which are made apparent by staining. However, for reasons that have been mentioned, the application of this result to the problem of human caries would be incorrect. A comparison of the teeth of Group III with those of Group III_c indicates that in the case of the lower teeth only is there a valid difference between the two groups. Thus it appears that the 20 p.p.m. of

TABLE IV

Total number of stained lesions observed under 15 × magnification

GROUP NUMBER	NUMBER OF ANIMALS	JAW	NUMBER OF LESIONS PER RAT	STANDARD DEVIATION (S_x)	RELATIVE DEVIATE (k)	PROBABILITY*
Comparison of Groups I and I_c II_c						
						per cent
I	26	Upper	3.23	2.24	−0.8	42.3
I_c II_c	46	Upper	3.76	2.10		
I	26	Lower	7.81	1.70	−0.7	48.3
I_c II_c	46	Lower	8.09	1.60		
Comparison of Groups II and I_c II_c						
II	42	Upper	2.60	2.00	−2.7	0.69
I_c II_c	46	Upper	3.76	2.10		
II	42	Lower	7.02	2.45	−2.5	1.24
I_c II_c	46	Lower	8.09	1.60		
Comparison of Groups III and III_c						
III	40	Upper	2.97	2.26	+1.4	16.1
III_c	37	Upper	2.35	1.65		
III	40	Lower	4.35	2.35	−3.2	0.14
III_c	37	Lower	5.89	1.87		

* Probability of difference of means arising from sampling errors.

fluorine given to these rats lowered the incidence of subsequent production of lesions capable of taking the Gomori stain. As has been mentioned, however, the result cannot be applied to conclusions relative to the probable effect of a similar treatment on the incidence of caries in human teeth.

In Table V are recorded the data showing the percentage of unfractured caries-susceptible areas which are affected by decalcification. By unfractured caries-susceptible areas is meant those of the 20 major fissures which were not associated with an obvious cusp fracture and in which a black staining could result only from the effects of the action of what is presumed to be a true carious process upon the

tooth structure. Thus the number of caries-susceptible areas may differ in each rat, varying inversely with the number of areas affected by fracture cavitation. The number of these caries-susceptible areas in each rat is obtained by subtracting

TABLE V

Percentage of caries susceptible areas (exclusive of fracture areas) affected by decalcification

GROUP NUMBER	NUMBER OF ANIMALS	JAW	NUMBER OF CARIES SUSCEPTIBLE AREAS			NUMBER OF CARIES			PER CENT OF CARIES	RELATIVE DEVIATE	PROBABILITY*
			Total for group	Mean per rat	S. D.	Total for group	Mean per rat	S. D.			
Comparison of Groups I and I_c II_c											
					($S_{\bar{x}}$)			($S_{\bar{x}}$)		(k)	*per cent*
I	26	Upper	241	9.27	1.05	79	3.04	2.12	32.8	−1.42	15.5
I_c II_c	46	Upper	448	9.74	0.78	171	3.72	2.10	38.2		
I	26	Lower	160	6.15	2.64	112	4.31	1.72	70.0	−1.74	8.2
I_c II_c	46	Lower	382	8.30	1.69	295	6.41	1.76	77.2		
I	26	Both	401	15.42	2.70	191	7.35	2.46	47.6	−2.80	0.51
I_c II_c	46	Both	830	18.04	2.02	466	10.13	3.08	56.1		
Comparison of Groups II and I_c II_c											
II	42	Upper	412	9.81	0.49	103	2.45	1.88	25.0	−4.18	<0.01
I_c II_c	46	Upper	448	9.74	0.78	171	3.72	2.10	38.2		
II	42	Lower	341	8.12	2.29	223	5.31	2.08	65.4	−3.51	0.04
I_c II_c	46	Lower	382	8.30	1.69	295	6.41	1.76	77.2		
II	42	Both	753	17.92	2.38	326	7.76	3.15	43.3	−5.06	<0.01
I_c II_c	46	Both	830	18.04	2.02	466	10.13	3.08	56.1		
Comparison of Groups III and III_c											
III	40	Upper	385	9.62	1.10	111	2.77	2.14	28.9	+1.79	7.3
III_c	37	Upper	366	9.89	0.37	85	2.30	1.66	23.2		
III	40	Lower	388	9.70	0.95	164	4.10	2.19	42.2	−4.19	<0.01
III_c	37	Lower	343	9.27	1.43	198	5.35	1.91	57.7		
III	40	Both	773	19.32	1.48	275	6.87	3.85	35.5	−1.73	8.3
III_c	37	Both	709	19.16	1.48	283	7.64	2.68	39.9		

* Probability of difference of means arising from sampling errors.

from 20 the number of major areas affected by fracture. By an area of decalcification is meant any lesion of the enamel of the unfractured caries-susceptible major fissures that can be detected because of the black color which it takes on during the staining process. These alterations of continuity of enamel, in our opinion, are the only lesions of rat molar teeth which are comparable to human

occlusal caries. These lesions thus constitute only the stained major areas which have not been affected by fracture.

Table V, based upon ratios or percentages, required different statistical treatment from that of Tables II, III, IV. The relative deviates (k) were calculated from the formulae given in, "Statistical Reasoning," by Treloar (pp. 207 & 208).

As has been mentioned, the data in Table V show the percentage of unfractured caries-susceptible areas in which decalcification has taken place. It is true that the effects of fractures are not completely eliminated by this method of treatment since the susceptibility to caries of the areas fractured may not be the same as that of the unfractured areas. However, if knowledge gained by a study of rat molar caries can be applied to human caries, it would seem that the results of the findings by this method would possess less error than would be the case for any of the methods employed by previous investigators of the effect of fluorine on rat molar caries.

A comparison of the teeth of Group I with those of Group I_e II_e indicates a very slight difference between the teeth of upper jaws of the two groups, a greater difference between the teeth of the lower jaws of the two groups, and a valid statistical difference when the upper and lower jaws are taken together. Thus it appears that the fluorine administered while the teeth were forming had some effect in lessening the incidence of caries. This effect was not great. However, it is statistically significant. It is possible that the high incidence of fracture lesions, especially in Group I and also in Group I_e II_e may have considerably reduced the apparent effect of the fluorine in reducing the caries attack rate since areas highly susceptible to caries were, in many instances, converted to fractures and for this reason were disregarded in the tabulation of true caries. It is interesting to compare the findings with respect to Group I as shown in Table V with those of Tables II and IV, which latter tables show primarily the fracture lesions. It can be observed that even though the total number of lesions of Group I is greater than of Group I_e II_e, the per cent of true caries in Group I is less than that of Group I_e II_e. This apparently indicates that although the fluorine which the young rats received made their teeth more liable to fracture and to gross destruction, it made them somewhat less susceptible to caries.

A comparison of the teeth of Group II with those of Group I_e II_e indicates a statistically significant lower incidence of caries in Group II for both upper and lower jaws and for the two jaws when taken together. This result would indicate that the fluorine which was supplied while the caries-producing diet was being fed lowered the caries incidence observed with respect to this group. This finding has been reported by a number of other investigators (4, 5, 6, 7, 8) who found the caries reduction to be much lower than was found in this investigation. These other investigators, however, used fluorine in concentrations much greater than was employed in the present study, and 3 of them (5, 7, 8) identified caries from lesions observed in unstained and unsectioned teeth. These variations in experimental methods perhaps account for the differences in the results.

A comparison of the teeth of Group III with those of Group III_e shows some in-

teresting results. The upper teeth of Group III showed slightly more caries than those of the control Group III_c, whereas the lower teeth of Group III showed markedly less caries than those of the control Group III_c. Thus it appears that the fluorine administered for 60 days and then discontinued brought about a decrease in the subsequent production of caries in the lower jaws. In the upper jaws, however, the fluorine treated group showed more caries. This seeming paradox can perhaps be explained by one or both of two possibilities. In the first place, the difference between the upper jaws of the two groups is not great enough to be of significance, whereas the difference between the lower jaws is unquestionably of valid significance. In the second place, the occlusal fissures of the upper molars are deeper and more narrow than those of the lower molars. This fact, plus location in the upper jaw, perhaps makes it more difficult for the fluorine-containing solutions to come into contact with the enamel in the depths of the fissures in the upper molars while still in a concentration great enough to bring about a significant change in the enamel composition.

The problem of cusp fracture offers a considerable obstacle in the evaluation of lesions in rat molar teeth and to the application of such findings to the study of true dental caries. It has been suggested that the cusp fractures in the rat molar teeth occur chiefly after the cusps have been undermined with caries and that therefore caries was the primary factor in bringing about these fractures. Our observations do not support this premise. Examinations of the teeth of rats (other than those used in this study) which had been on a coarse cornmeal diet have disclosed that fracture-type lesions began to occur almost immediately, although obviously, the longer the period the animals are fed this type of diet, the more numerous and the more extensive these lesions will become. It is unfortunate that the rat, which is in so many ways suitable for caries studies, has molar teeth which are so susceptible to fracture; or perhaps we should say, it is unfortunate that a diet which brings about fractures must be used for caries production in the rat. There can be little doubt that most or all studies of caries in the rat have been influenced in one way or another by these fractures. The devising of a caries-producing diet for rats which would not fracture their teeth or the finding of a new animal (22) whose teeth would readily develop caries on a diet, regardless of the physical properties of this diet, is a much needed forward step in the study of dental caries.

SUMMARY AND CONCLUSIONS

1. This investigation was carried on to gain further information as to the effect of fluorine upon experimental dental caries in the rat. Several methods for scoring the dental lesions were used; the conclusions of this investigation were based upon a scoring method not heretofore used in fluorine—dental caries studies.

2. The incidence of dental fractures and dental caries was studied when rats were fed extra-dietary fluorine (1) during formation of the molar teeth, (2) simultaneously with the caries-producing diet, and (3) after molar eruption but previous to the regimen on the caries-producing diet.

3. The molar teeth of rats fed a coarse cornmeal diet for 10 weeks, beginning at

age 45 days, showed more fractures than those of animals fed the same diet for the same length of time beginning at age 110 days.

4. The molar teeth of rats whose mothers received, during pregnancy and lactation, drinking water containing 20 p.p.m. of fluorine and which rats were given daily for the first 21 days of life, 0.1 mg. fluorine, were rendered more liable to fracture as shown when fed the coarse cornmeal diet beginning at age 45 days. This alteration in the quality of the teeth may have been a result of a change in their structure consequent upon an inadvertent overtreatment of the animals with fluoride.

5. In 41 per cent of the 26 animals mentioned in item 4 above, the mesio-buccal cusp of the upper third molar was missing. In none of the other 165 animals employed in this study was this cusp found to be absent.

6. Extra-dietary fluorine, to the extent of 20 p.p.m. in the drinking water, had no influence on the incidence of fracture lesions of rat molar teeth when administered to the animals either simultaneously with or previous to the regimen on the coarse cornmeal diet.

7. The molar teeth of rats whose mothers received, during pregnancy and lactation, drinking water containing 20 p.p.m. of fluorine and which rats were given daily for the first 21 days of life, 0.1 mg fluorine, were rendered slightly more resistant to cornmeal caries.

8. The caries susceptibility of rat molar teeth was decreased by supplying drinking water containing 20 p.p.m. of fluorine simultaneously with the caries-producing diet.

9. The caries susceptibility of the lower molar teeth of rats was decreased when the animals were supplied with drinking water containing 20 p.p.m. of fluorine for a period of 60 days before the regimen on the caries-producing diet during which they received no added fluorine.

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THE MECHANISM BY WHICH IODOACETIC ACID INHIBITS EXPERIMENTAL DENTAL CARIES IN THE RAT[1]

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The production of carious lesions in rats maintained on a caries-producing diet is inhibited when the ration is supplemented with 200 p.p.m. (parts per million) of iodoacetic (iodoethanoic) acid in the food, 20 p.p.m. in the drinking water, or both (1). The mechanism by which the substance exerts its effect has not been adequately described (2, 3). This study was conducted in an attempt to ascertain whether the caries-inhibiting action of iodoacetic acid is regional or systemic.

EXPERIMENTAL

Experimental methods applied to the study of the process by which fluorine limits rat caries were employed to determine the mode of action of iodoacetic acid. These are desalivation of the animal to test whether the effect is mediated locally through the saliva (4) and parenteral administration to demonstrate a possible systemic mechanism. Topical applications of the drug to the teeth in situ (5) in experimental animals are now in progress to examine directly the potentiality of regional effectiveness.

Fifty-seven female albino rats of pure inbred stock weighing 70 to 80 grams each were subjected to investigation. Litter mates were divided into 5 groups, all maintained on the Hoppert-Webber-Canniff (6) coarse corn diet throughout the experimental period of 150 days. Group I, 13 normal rats, served as control, the animals receiving no iodoacetic acid. Group II, 11 desalivated animals, received no iodoacetic acid. Group III, 12 non-desalivated rats, received subcutaneous injections of 1 cc. of a sodium iodoacetate solution every second day. The solution consisted of normal saline containing 400 p.p.m. of iodoacetate buffered with NaOH to pH 7.2. Group IV, 11 unoperated rats, received the basic ration plus 200 p.p.m. of iodoacetic acid in the drinking water. Group V, 10 desalivated animals, received a supplement of 200 p.p.m. of the substance in the food. All animals were fed food and water ad libitum and their weights recorded weekly.

On completion of the experiment, the rats were sacrificed, the jaws removed,

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and the molar teeth carefully examined for caries under a binocular (×10) microscope. Lesions were scored (1) by three examiners using a modification of the

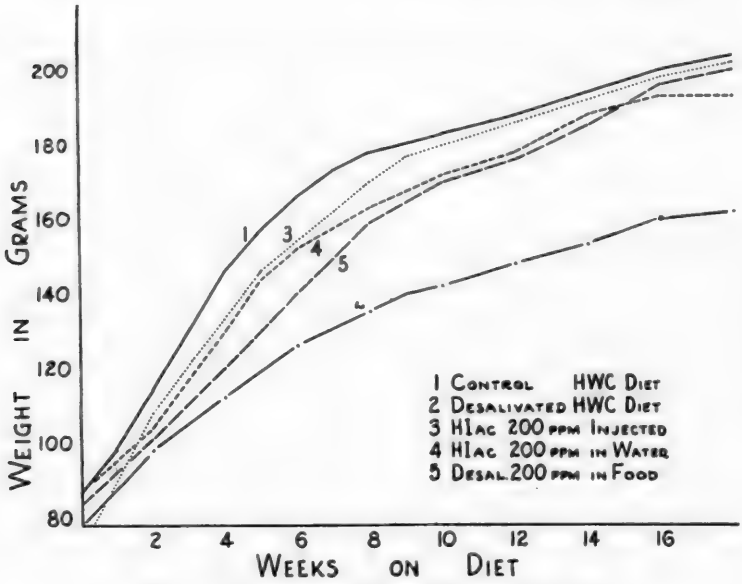

FIG. 1. Average growth of experimental animals

TABLE I

GROUP	NO. OF ANIMALS	NO. OF CARIES-FREE ANIMALS	NO. OF TEETH EXAMINED	NO. OF CAVITIES	NO. OF AREAS AFFECTED	SUM OF TOTAL SCORES	AVERAGE CARIES SCORE PER ANIMAL
I Control. H.W.C.* and H_2O	13	1	156	31	70	184	14.1
II Desalivated. H.W.C.* and H_2O	11	0	132	39	106	289	26.3
III Non-desalivated. 1 cc. 400 p.p.m. HIac injected every 2nd day, H.W.C.* and H_2O	12	6	144	11	14	27	2.2
IV Non-desalivated. 200 p.p.m. HIac in H_2O, H.W.C.* and H_2O	11	4	132	9	14	33	3.0
V Desalivated. 200 p.p.m. HIac infood, H.W.C.* and H_2O	10	6	120	9	17	35	3.5

* Hoppert-Webber-Canniff coarse corn diet.

method of Cox, Dodds, Dixon and Matuschak (7). Individual results were not significantly different.

RESULTS

Average weight curves for the animals in the various experimental groups are shown in *fig. 1*. No change from normal growth was observed, excepting in desalivated rats which received no iodoacetic acid (group II). At the end of the experiment, these averaged 165 grams each, while desalivated animals receiving a supplement of 200 p.p.m. of iodoacetic acid in the food (group V) averaged 204 grams each. Rusting (8) appeared in all the desalivated rats 6 weeks post-operatively, and became more pronounced as the experiment progressed.

The animal groups, their diets, and the caries scores are recorded in Table I. A marked increase in the incidence and severity of dental lesions was found in rats which did not receive iodoacetic acid (groups I, II) as compared with those which did (III, IV, V). Desalivated rats on the basic ration only (II) exhibited an average caries score per animal 85 per cent greater than the control rats (I). The differences between the scores obtained for animals which received iodoacetic acid in the food, in the water, and parenterally (III, IV, V) are not significant.

DISCUSSION

The results substantiate previous reports (1, 2, 3) that iodoacetic acid supplements in the food and drinking water inhibit experimental dental caries in the rat. They also verify the finding of Cheyne (9) that extirpation of the salivary glands results in a higher incidence of caries in rats fed a coarse corn diet. The observation that iodoacetic acid was apparently responsible for normal growth in the desalivated animals which received it (group V) suggests a possible growth-stimulative effect of the drug when administered in non-toxic doses.

Unlike fluorine (10), parenteral administration of iodoacetic acid was capable of reducing the incidence and severity of dental lesions in rats, indicating that iodoacetic acid may exert its influence systemically. The feasibility that it is secreted in the saliva appears to be ruled out by the finding that the substance was as effective in diminishing caries activity in desalivated animals as it was in unoperated rats. Nevertheless, to establish the possible systemic nature of the action of the drug, its action when injected into desalivated animals should be investigated. Since iodoacetic acid administered orally or parenterally seems to be equally efficacious in controlling experimental caries, it is likely that the amount administered rather than the mode of administration is a deciding factor in determining its effectiveness. Evidence substantiating this impression has been reported (1).

SUMMARY

Experimental dental caries in the rat is inhibited by parenteral administration of iodoacetic acid to a degree comparable with its effectiveness when administered in the food or drinking water. Since iodoacetic acid diminishes caries activity in desalivated as well as normal animals, it appears that the mechanism of its action may be systemic.

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EFFECT OF PREGNANCY ON THE CHEMICAL COMPOSITION OF HUMAN DENTIN[1]

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The effect of pregnancy upon the teeth has been the subject of much interest in the past. Karlstrom (11) reported that the percentage of ash, calcium and phosphorus of the incisor teeth of rats after delivery did not differ from those of control animals. He concluded that his findings did not confirm the popular idea that the chemical composition of the teeth may undergo a change during pregnancy. It should be noted that the animals were fed a mixed diet, which probably contained sufficient amounts of all the necessary food elements. Toverud (18) made a similar study but kept the rats, during the gestation period, on a diet that was deficient in calcium and vitamin D. He found that the percentage of ash, calcium and phosphorus of the incisor teeth progressively decreased as a result of successive pregnancies. The values obtained for the animals carried through pregnancy were markedly lower than those of the non-pregnant controls kept on a similar diet. Rosebury and Foley (14) reported a study in which the pregnant rats were fed a diet deficient in calcium and vitamin D. They found by histological examination that the incisors and molars were defectively calcified. The incisors were affected to a greater degree than the molars. The addition of an adequate amount of vitamin D to this diet did not completely prevent defective calcification of the teeth. In connection with these studies it should be noted that, instead of withdrawal of calcium and phosphorus, the result of pregnancy may have been to decrease the rate of deposition of these elements in the teeth. Furthermore, the changes in the composition of the incisors of rats may not be applicable to the fully formed teeth of humans, since the incisors of rats are teeth of persistent growth.

An analysis of the reports based on clinical observation reveals a difference in the conclusions regarding the relationship between pregnancy and the teeth. Ziskin (20) in a study of 599 pregnant women and 205 non-pregnant women found that the former group, examined during the late months of pregnancy, had fewer decayed teeth than the latter group. In a study by Ziskin and Hotelling (21), it was shown that pregnancy not only does not increase tooth decay but that factors operating during pregnancy actually prevent tooth decay to a significant degree. Starobinsky (17) studied the incidence of dental caries in 150 non-pregnant women and in 216 pregnant women at the ninth month of pregnancy

[1] Read at a meeting of the William Jarvie Society of the School of Dental and Oral Surgery, Columbia University, February 19, 1943. Received for publication May 27, 1943.

and found that there was an increase in the number of decayed teeth with advancing age, in pregnant as well as the non-pregnant women. The increase in both groups was about the same, the non-pregnant group showing a slightly larger number of decayed teeth than the pregnant. However, Gerson (7), from a study of 50 pregnant and 50 non-pregnant individuals, suggested that a positive relationship exists between caries and pregnancy in humans.

Many of the reports on the relationship of pregnancy and teeth offer no evidence, either experimental or clinical. They reveal a widespread acceptance of the clinical impression that pregnancy induces an increased tendency to tooth decay (1, 2, 3, 4, 5, 8, 9, 10, 13, 16, 19).

In the present study, an attempt was made to ascertain whether pregnancy has any effect on the ash, calcium and phosphorus contents of the dentin of human teeth.

METHOD

The teeth were extracted during the course of dental treatment. Thirty-one teeth were extracted from pregnant Vanderbilt Clinic patients. No attempt at selection of cases was made in this group. The teeth of the control group, 21 in number, were from females of approximately the same age range.

The teeth were placed in 95% alcohol immediately after extraction. Before analysis all adherent soft tissue, blood and carious material were removed. The teeth were washed in water and allowed to dry in air at room temperature. The anatomical crown of each tooth was then removed, using "lightning discs", and the roots denuded of cementum with a No. 11 stone. The root dentin was then split in a steel mortar, the dried pulp removed, and the dentin crushed to a coarse powder. The lipids of the dentin powder were extracted for 3 hours in a Soxhlet apparatus with a 1:1 mixture of ethyl alcohol (95%), and ether, which was made alkaline to phenolphthalein with 1% NaOH. The alcohol-ether was removed by allowing the powder to remain exposed to the air, at room temperature, for a minimum of 24 hours. The material was dried at 105° C for 24 hours and transferred to a desiccator. Weighed samples (approximately 50 mg.) of each specimen were heated at 650° C. in a porcelain crucible for 48 hours. The weighed ash was dissolved in 10 cc. of 1 N HCl, transferred to a 25 cc. volumetric flask and diluted to mark. Aliquots of this solution were used for determinations of calcium and phosphorus.

The method used for the determination of the calcium is a modification of the Shear and Kramer method (15). A modification of the Fiske and Subbarrow method (6) was used for the phosphorus determination.

Calcium Determination: To a 5 cc. aliquot in a 25 cc. centrifuge tube are added 4 drops of bromcresol purple and 6 cc. of 4% ammonium oxalate (there should be at least 1 cc. of ammonium oxalate for approximately 1 mg. of Ca in the aliquot). The pH of the mixture is brought to about 6.0 (lavender color), starting with 1 N NH_4OH, and using 0.2 N NH_4OH, 0.5 N HCl, and 0.1 N HCl for final adjustment. The mixture is allowed to stand over night for complete precipitation of calcium oxalate and centrifuged for 10 minutes. The supernatant liquid is poured off and the tube placed in an inverted position and allowed to drain for 5 minutes. The precipitate is then broken up with a glass rod, and mixed with 1.5 cc. of

acetone.[2] Then 3 cc. of 0.2 N ammonium hydroxide is mixed with the acetone. Another 3 cc. of NH_4OH is added to the surface without agitation, the sides of the tube being washed during the addition. On top of this layer of NH_4OH is added 0.5 cc. of acetone, without mixing. The whole is centrifuged again for 10 minutes, the supernatant liquid poured off,

TABLE I

Analysis of dentin of non-pregnant (control) group

SPECIMEN NUMBER	AGE OF SUBJECT	NO. OF PREVIOUS PREGNANCIES	CALCIUM	PHOSPHORUS	Ca/P	ASH	TOOTH* ANALYZED	REASON FOR EXTRACTION**
			per cent	*per cent*		*per cent*		
2	26	1	26.0	12.5	2.08		8	A
3	35	4	26.3	12.5	2.10		7	J
6	32	2	26.2	13.1	2.01		1	J
9	35	4	26.4	13.0	2.03		5	A
10	40	8	26.2	12.9	2.03		5	A
11	34	2	26.4	12.8	2.06		4	A
12	27	2	27.2	13.2	2.07		6	A
16	32	10	26.3	12.7	2.07	68.5	6	C
19	35	3	27.4	12.8	2.14	68.2	5	A
23	32	10	26.5	12.7	2.08	70.0	7	C
28	30	2	26.7	12.3	2.18	71.4	6	A
30	38	2	26.1	12.4	2.11	74.4	7	A
33	38	3	25.7	12.3	2.09	71.3	4	A
35	38	2	26.9	12.5	2.16	71.4	6	A
40	38	2	27.0	13.1	2.05	69.4	6	A
42	38	2	27.6	12.6	2.11	70.9	7	A
46	38	2	25.8	12.8	2.02	71.9	5	A
47	34	1	26.7	12.6	2.11	71.4	5	A
49	31	4	26.5	12.9	2.06	70.8	2	C
50	26	2	26.0	12.5	2.08	70.9	8	A
51	38	2	26.3	12.7	2.08	69.3	8	A
Mean	34	3	26.5	12.7	2.08	70.7		

* 1 refers to first incisor; 2—second incisor; 4—first premolar; 5—second premolar; 6—first molar; 7—second molar; 8—third molar.

** A refers to caries with pulp involvement; C—pulpless and evidence of periapical infection; J—advanced periodontoclasia.

and the tube allowed to drain for five minutes. The washing is repeated but the precipitate is not broken up. Following the second washing, the acetone is allowed to evaporate spontaneously for 24 hours. The precipitate is then dissolved in 5 cc. of 2 N H_2SO_4, heated in a water bath (100° C.) and titrated with 0.025 N $KMnO_4$. The permanganate was standardized with 5 cc. of 0.02 N Na oxalate[3] to which was added 5 cc. of 2 N H_2SO_4.

[2] Acetone was added to prevent calcium oxalate crystals from remaining on the surface of the supernatant fluid after centrifuging (12).

[3] The sodium oxalate standard should be kept in an amber bottle to prevent decomposition.

TABLE II

Analysis of dentin of pregnant group

SPECIMEN NO.	AGE OF SUBJECT	NO. OF PREVIOUS PREGNANCIES	MONTH OF PREGNANCY	CALCIUM	PHOSPHORUS	Ca/P	ASH	TOOTH* ANALYZED	REASON FOR EXTRACTION**
				per cent	*per cent*		*per cent*		
1	30	2	9	26.2	12.4	2.13		7	A
4	28	1	6	26.8	12.7	2.11		7	J
5	31	0	5	26.3	13.0	2.03		7	J
7	28	1	6	25.5	12.5	2.03		6	A
8	26	2	4	26.8	12.8	2.09		5	A
13	28	1	6	27.9	13.4	2.08	70.0	3	C
14	31	3	7	26.3	13.0	2.02	72.3	6	A
15	28	1	6	26.8	13.1	2.05	69.6	7	J
17	26	2	4	25.3	13.1	2.01	66.8	5	A
18	22	1	8	27.5	13.0	2.12	68.5	7	C
20	23	2	6	26.4	12.9	2.06	68.8	4	A
21	29	2	4	26.6	13.0	2.05	70.4	7	C
22	32	0	3	26.3	12.7	2.08	69.7	8	F
24	23	2	6	25.9	12.8	2.03	68.8	5	J
25	22	2	5	26.3	12.7	2.07	69.3	5	C
26	25	1	4	26.6	12.7	2.09	70.0	6	A
27	20	0	7	26.2	12.7	2.07	69.4	4	A
29	35	6	8	26.1	12.3	2.12	74.9	6	E
31	35	6	8	27.2	12.9	2.12	71.1	5	E
32	29	2	5	26.9	12.8	2.10	70.3	5	A
34	35	6	8	25.4	12.4	2.05	71.9	7	E
36	20	0	7	25.7	12.5	2.05	70.5	5	A
37	32	0	3	26.7	12.6	2.11	71.1	6	A
38	29	2	5	27.2	12.1	2.25	70.0	6	H
39	33	5	6	26.9	12.8	2.10	68.9	7	A
41	25	0	5	26.0	12.3	2.11	71.2	8	A
43	22	0	6	26.4	12.5	2.10	70.4	8	A
44	29	2	5	26.1	12.9	2.03	69.8	4	E
45	29	2	7	26.8	12.4	2.16	72.2	5	A
48	35	6	8	26.7	12.5	2.14	71.0	5	E
52	26	0	9	26.4	12.6	2.10	71.1	8	A
Mean	28	2	6	26.5	12.7	2.08	70.3		

* 3 refers to canine; 4—first premolar; 5—second premolar; 6—first molar; 7—second molar; 8—third molar.

** A refers to caries with pulp involvement; C—pulpless and evidence of periapical infection; E—only root left; F—impacted; H—useless; J—advanced periodontoclasia.

Phosphorus Determination: A 3 cc. aliquot of the dentin solution is transerred to a 50 cc. volumetric flask. Add 10 cc. of water and 10 cc. of molybdate solution (2.5% ammonium molybdate in 5 N sulphuric acid) and mix. The neck of the flask is washed with 10 cc. of

water to prevent direct mixing of ammonium molybdate and reagent which follows. Finally, 4 cc. of sulphonic acid reagent is mixed in; the solution is diluted to mark and compared with standard after the color has been allowed to develop for 10 minutes. The standard is prepared by adding 10 cc. of standard phosphorus solution (10 cc. = 0.8 mg. P) to a 50 cc. volumetric flask, and proceeding as with the unknown.

RESULTS

The results of the analyses are presented in Tables I and II.

It is evident from the results obtained for the calcium, phosphorus and ash contents of the dentin that no difference was found between the non-pregnant and pregnant groups of women. When the teeth were grouped according to month of pregnancy, number of previous pregnancies and age of the individual, no differences were found between the two groups. It appears, therefore, that the percentages of calcium, phosphorus and ash of the root dentin of humans are not influenced by pregnancy. This indicates that the belief that minerals are withdrawn from the teeth during pregnancy is not valid.

SUMMARY

Root dentin of 31 teeth of pregnant women, and 21 teeth of non-pregnant women were analyzed for percentages of ash, calcium, and phosphorus. No difference was found between the groups. This indicates that there is no basis for the view that minerals are withdrawn from the teeth of humans during pregnancy.

The authors wish to thank the Divisions of Oral Diagnosis and Oral Surgery for their cooperation in obtaining the teeth for analysis.

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ANTHROPOLOGICAL STUDIES IN DENTAL CARIES[1]

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The great prevalence of dental caries among modern civilized peoples is a matter of record. The disease begins early in life, and statistical studies have established the unwelcome fact that almost every school child is subject to it. It is also known that ancient peoples and primitive races suffered less from this ailment. However, investigations here have not been as thorough. Statistical studies have not been made.

As a result, many leading scientists tend to express extreme views on this subject. Thus, only recently, Merritt (1), stated as follows: "No one knows the cause of the two most universal dental ailments, namely, tooth decay and pyorrhea. . . . It is known that primitive man suffered from neither of these affections." A contrary view is presented by Weinberger (2), who states: "That we are dealing with new problems, generally believed to be the result of modified conditions brought about through modern civilization, is not a fact, for ancient man, physically, in some respects, was very much as man of today. There is sufficient evidence to show that he suffered from caries as well as abnormalities in the position and irregularities of the teeth, and that unerupted, supernumerary, retained and impacted teeth date back to the earliest remains of the human type." Evidently, such contradictory and irreconcilable statements by two dental scientists not only fail to clarify matters, but tend to further befog the issue, and leave us floundering in a mist of uncertainty on a very important subject.

What is the true status of caries in primitive man? Was he immune or partly immune to the disease? If so, what was the mechanism of this immunity? To find appropriate answers to these questions, we have gone directly to the original subject matter. We have undertaken anthropological studies of dental caries in skull collections of various racial stocks. Our findings are presented in this paper.

OBJECT AND METHOD

The anthropological division of the American Museum of Natural History in New York contains one of the richest skull collections in the world. Various races and nationalities, and peoples of all continents and diverse eras find representation in this collection. Those skulls that were unearthed in the same geographical area are grouped together. Our study includes 46 of these geographical groups containing approximately 4,000 skulls.

[1] Presented at a meeting of the New York Section of the International Association for Dental Research, December 28, 1942, and by title, at the 21st General Meeting of the I.A.D.R., Chicago, March 13 and 14, 1943 (*J. D. Res.*, **22**: 225, 1943). Received for publication March 30, 1943, revised April 22, 1943.

TABLE I

Caries incidence in 46 geographic groups

NAME OF GEOGRAPHIC GROUP	NO. OF SKULLS	NO. OF TEETH	NO. OF CARIOUS TEETH	PERCENTAGE OF CARIOUS TEETH
New Zealand	49	399	9	2.2
Solomon Islands	32	139	1	0.7
Tahiti	35	145	2	1.3
Easter Island	43	235	9	3.8
Hawaii	12	188	—	—
Chatham Island	21	199	—	—
New Britain, Ralum	130	1859	8	0.4
New Guinea	30	281	1	0.3
Egypt, Giseh	69	356	14	3.9
Egypt, Nubia	10	58	2	3.4
Egypt, El Hessa	137	1805	82	4.5
West Africa	20	364	4	1.1
South Africa	35	732	7	0.9
China	53	464	24	5.0
India	91	1901	33	1.7
Japan & Korea	23	362	10	2.7
Malay Peninsula	91	450	20	4.4
Mongolia	22	78	4	5.1
Siam	36	278	19	6.8
Siberia	10	76	—	—
Asia Minor	269	1211	52	4.3
Hungary	178	2446	216	8.8
Italy	14	130	11	8.4
Yugo-Slavia	68	109	37	34.0
Poland	15	87	8	9.1
Russia	37	306	11	3.5
Greece	108	378	24	6.3
Germany	91	745	164	22.0
Austria	166	692	64	9.2
Honduras	10	143	8	5.5
Porto Rico	28	544	23	4.2
Venezuela	14	176	17	9.6
Peru	232	1867	152	8.1
Colombia	12	230	6	2.6
Bolivia	135	659	17	2.6
Chile	26	363	14	3.8
Patagonia	27	125	—	—
Mexico	346	3298	153	4.6
Eskimo	255	1974	—	—
British Columbia	337	3077	—	—
Oregon	61	444	5	1.1
Washington	182	1979	18	0.9
Utah	148	1690	77	4.6
New Mexico	208	3246	127	3.9
California	30	438	7	1.6
Alaska & Aleutians	45	565	2	0.4

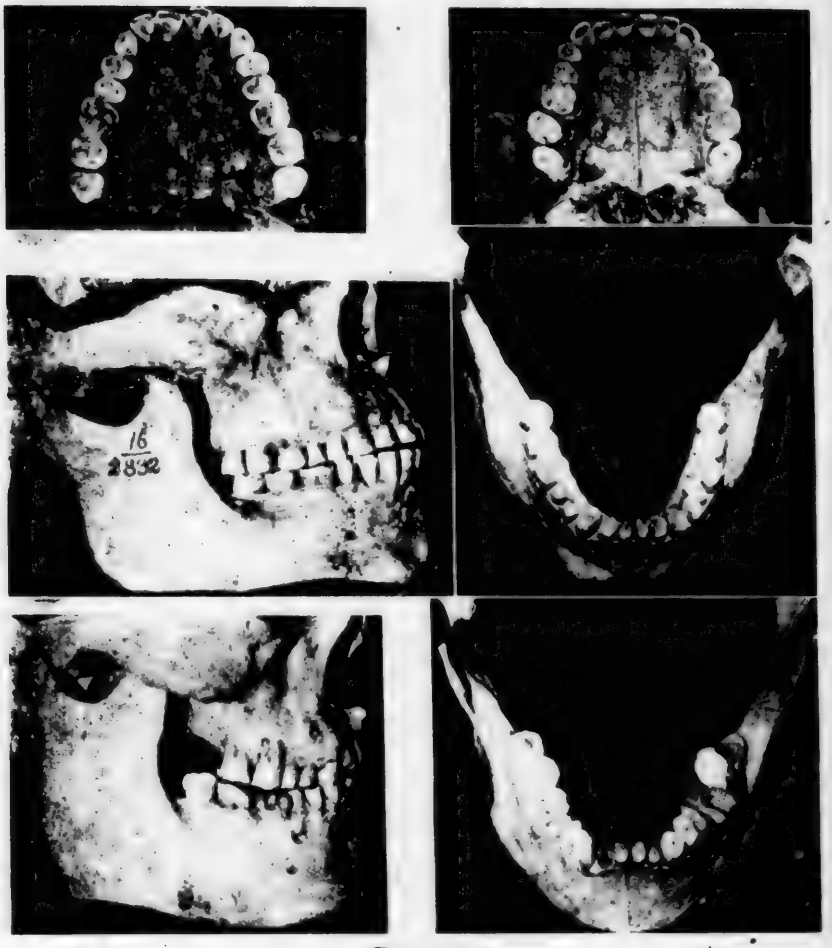
FIGS. 1-4

FIGS. 1a (upper left) & 1b (upper right). El Hessa, Egypt, #3111. Anterior and lateral ews of ancient Egyptian skull, demonstrating features observed in most primitive jaws. ete full complement of teeth, well articulated and in functional stage of attrition. Promi- nt zygomatic arches and rugged character of maxillary and mandibular bones bespeak werful and vigorous function.

FIGS. 2a (middle left) & 2b (middle right). Siberia, #7777. Anterior and lateral views deciduous dentition in primitive 3 year old child. Note prominent zygomatic arch, ing ramus, and rugged angle of mandible where masseter muscle attaches.

FIG. 3 (lower left). Russia, #3591. Palatal view, showing caries only on third molars, ich have functioned least.

FIG. 4 (lower right). New Britain, #1518. Palatal view, showing all teeth well worn and e of dental caries, except for malposed right second incisor and canine. Extensive decay e is due to local food retention and stagnation.



FIGS. 5–8

FIG. 5 (upper left). Washington, #22171. Palate of American Indian, with teeth showing advanced physiologic attrition.

FIG. 6 (upper right). Eskimo, #100. Sturdy, square palate, with transitory stage of attrition registered on all teeth except third molars.

FIGS. 7a (middle left) & 7b (middle right). British Columbia, #2832. Lateral view of articulated skull and anterior view of mandible, showing transitory stage of attrition, with obliteration of occlusal anatomy and wear extending well into dentin.

FIGS. 8a (lower left) & 8b (lower right). New Zealand, #783. Lateral view of articulated skull and anterior view of mandible, showing teeth in senile stage of attrition, with wear past proximal contacts and exposure of pulps, despite the laying down of secondary dentin. Note periapical abscesses at L.R.5 and U.R.4.

These photographs were taken with the kind permission of the American Museum of Natural History, New York, N. Y.

Our first objective was to establish statistically the incidence of caries within each of these geographical groups. We counted the number of teeth remaining in each skull, and carefully examined each tooth for caries. Then the percentage of carious teeth was computed for the group. Many teeth were found to have been broken or lost from the jaws after death. In establishing the caries index of a live subject, filled, crowned, and missing teeth are usually attributed to an advanced carious lesion, and a factor is added to the total number of carious surfaces. Because of the factor of posthumous damage to the skulls, it was decided to ignore all missing and fractured teeth in the count. Only the 38,300 unbroken teeth actually found in or with the skulls were included in the statistical studies. No dental restorations were found in the ancient and primitive groups studied.

Our second object was to search for possible reasons why certain individual skulls or groups of skulls were more susceptible to dental caries than others. Special attention was paid to the state of development of the jaw bones and the various points which serve as the origin and insertion for the muscles of mastication. The shape of the palate was noted; the anatomy of individual teeth was studied, with particular attention paid to the presence and extent of attrition.

A statistical summary of our findings is given in Table I.

STATISTICAL FINDINGS

Examination of Table I reveals that, of the 46 groups examined, 6 were completely caries free. The Eskimo group, with 255 skulls and 1974 teeth, and British Columbia, with 337 skulls and 3077 teeth, are fairly large collections and are representative enough to warrant the conclusion that these peoples were immune to caries. The other four groups—Hawaii with 12 skulls and 188 teeth; Chatham Island, 21 skulls and 199 teeth; Siberia, 10 skulls and 76 teeth; and Patagonia, 27 skulls and 125 teeth—are too small to be representative. All of the 40 other groups showed various degrees of caries, ranging from 0.3% in the New Guinea group to 34% in the Yugo-Slav group.

The most ancient skulls represented in the collection came from Egypt. Of these, there was 3.9% caries incidence in the Giseh group; 3.4% in Nubia; and 4.5% in El Hessa. The relatively high state of civilization achieved in ancient Egypt may be a consideration here.

The general tendency in the groups studied was for a lower incidence of caries in the ancient and primitive peoples, increasing in the modern and civilized groups. All primitive peoples were not immune to caries.

DISCUSSION

The fundamental chain of factors required to initiate a carious lesion is as follows: Acid forming microorganisms collect in sheltered areas, such as the interproximal spaces or occlusal pits and fissures. They are protected from the saliva by plaque formations, and are nourished by carbohydrate food residues. Their metabolic products are acids which dissolve away tooth structure. In order to prevent caries, this chain must be broken. We believe that this is best

accomplished by proper mastication. The teeth of the primitives needed no artificial methods of cleansing. The mouths of these peoples were naturally clean. The anatomy of sound, healthy teeth and soft tissues is such that food particles could not adhere and stagnate. Gingival tissues of good tone and contour probably completely filled the interproximal spaces up to the firm proximal contacts. Natural, coarse, fibrous, detergent foods, vigorously chewed, swept clear the fissures and sluiceways of occlusal anatomy. It was the forceful and thorough mastication of detergent foods that gave primitive groups their caries freedom.

With the advance of civilization, the human brain has devised mechanical aids and refinements to ease the physical tasks of the body. Important in this respect is the development of the culinary art. Modern foods are cooked, boiled, baked, cut, chopped, and mashed into sophisticated forms. Their soft, sticky consistency causes them to adhere to the teeth. They lack the coarse particles and rough fibers which cleanse the teeth during mastication and lack the ability to stimulate more than a minimum of masticatory function.

Biologists know that it is function that determines structure. Thus, the degree of masticatory function is reflected in the anatomy and contour of the bones which serve as origin and insertion for the muscles of mastication. In a primitive skull that has developed with vigorous masticatory function, prominent zygomatic arch, a square, heavy mandible with forceful ridges and broad sulci for muscle attachments are found. The palate is broad and square, the alveolar process rugged, and the occlusion even. The teeth also show the marks of thorough, powerful functioning. A physiologic state of attrition has been achieved, and dental caries is minimal or absent.

The importance of lack of masticatory function as a cause for caries is emphasized by our observation of some skulls in which all teeth were caries free with the exception of the third molars. The latter teeth showed no attrition, since they were not quite reached by the food and functioned less than the rest. Another example is where malposed teeth created a food trap in which an advanced carious lesion developed. There is nothing mysterious about the caries freedom of the other teeth in those jaws. They were not immune by virtue of complex antigen-antibody reaction. They were merely properly aligned and kept mechanically clean by proper function.

That the masticatory apparatus of primitive man functioned better than that of modern man is attested to not only by the general anatomy of the skulls but also by the degree of attrition registered on their teeth. Attrition is the gradual wearing away of the hard structure of the tooth through the physical and physiologic agencies of food mastication. During function, the morsal surfaces of the teeth gradually yield some of their substance to the friction of the food and of one surface against the other. Attrition is a slow process, constantly advancing with age.

On a previous occasion one of us (M. K.) dealt with this subject in more detail. We pointed out that dental attrition is not a pathological but a physiological phenomenon, resulting from the natural functional process of food mastica-

tion (3). Attrition was divided into three stages: the physiologic stage, where only the enamel shows wear; the transitory stage, when a substantial amount of dentin is lost; and the senile stage, when the proximal contacts are lost and the pulps are exposed and devitalized. The rapidity of the course of attrition depends mainly upon the amount of function to which the teeth are subjected in the process of mastication, and upon the physical nature of the food consumed. Hard, bulky, resistant foods, which demand greater pressure and exercise of the masticatory apparatus, wear the teeth more rapidly than liquid, soft, resistless foods. Very little attrition is found in modern, civilized man; but attrition is characteristic of the teeth of ancient, uncivilized and primitive peoples. In the latter, dental attrition starts early in life. Even the teeth of young subjects show distinct evidence of wear.

In the physiologic stage of attrition, the teeth and supporting structures are in a perfectly healthy state, showing no caries or other ailments. The senile stage, on the other hand, is usually accompanied by bone destruction, periapical osteitis, peridontosis, and root caries. Caries, at this stage, may be induced by the wedging of food between the teeth due to the loss of proximal contacts.

There can be no attrition without function. Therefore attrition is the index of function; it is the yardstick by which masticatory function can be measured. In the examination of skull collections, attrition serves as a guide in determining the degree in which the masticatory apparatus functioned during the life of each individual. Our investigation shows an inverse proportion between masticatory function and the incidence of dental caries.

At the present moment, when the incidence of caries is assuming larger and more alarming proportions among modern, civilized communities, we must pose the very pertinent question: Is it lack of function of the masticatory apparatus which is mainly responsible for this deplorable condition? Our answer is in the affirmative.

SUMMARY AND CONCLUSION

The incidence of dental caries among various peoples of diverse periods was investigated. 4,000 skulls and 38,300 teeth were examined. Of the 46 geographical groups, 6 showed no caries. The more primitive groups tended to have a lower incidence of dental decay than the more modern and civilized peoples. Anatomic studies of the masticatory apparatus of all the groups show that the jaw bones of the primitives were better developed and the teeth evinced greater attrition than those of modern peoples. This attests to the higher degree of function of the masticatory apparatus of these races. There is an inverse proportion between masticatory function and the incidence of dental caries. Lack of dental function is an important factor in the causation of caries among modern peoples.

We are grateful to Dr. H. L. Shapiro, Curator, and Miss Bella Weitzner, Associate Curator of the American Museum of Natural History for the kind assistance and invaluable advice which made these studies possible

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EFFECT OF AGE ON THE INCIDENCE OF EXPERIMENTAL CARIES IN NORMAL AND CASTRATE FEMALE RATS[1]

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Attention has recently been focused on the problems of aging of dental structures by Robinson, Boling and Lischer (1). Although these authors have compiled an imposing list of references concerning the changes in the teeth and jaws with age, little is known of the effect of age on experimental caries in the rat. Most of the studies on caries in rats (2–19) have been made over relatively short periods and on young animals. Only a few studies (20–26) have included rats of advanced age or rats which have been observed over a period of 1 year or more. From these long-term studies the general impression is gained that rats on adequate dietary regimes do not develop caries, whereas those fed deficient diets may have carious teeth. No data are available on the incidence of caries in old rats placed on the coarse corn diet.

A colony of 17 normal and 12 castrate female rats were put on the Hoppert, Weber, and Caniff diet (3) at the age of 1 year. They were maintained on this diet until death from old age or other causes supervened, with the exception of 4 rats which were sacrificed after 19 months on the diet in order to end the experiment. At death the jaws were removed and the teeth examined under binoculars (15×) according to the technique previously described (25). Missing cusps and microscopic lesions were recorded although it was frequently difficult to distinguish between fractures and carious lesions in the presence of advanced attrition.

The data on the normal rats are given in Table I. It is interesting to observe that little evidence of caries was found in some of the rats after periods as long as 13 months on the experimental diet. The number of cavities, the number of cusps involved, and the number of teeth totally destroyed are seen to increase with increasing time on the diet. In Table II are given the corresponding data on the castrate female rats. None of these had any caries up to 9 months on the diet. As in the case of the normal animals the numbers of cavities, of cusps involved, and of teeth totally destroyed increased with the time on the diet.

There is little difference between the observations on the normal and on the castrate female rats. The normal rats averaged 2.8 cavities per rat; the castrates 2.2. The normal rats averaged 7.5 cusps involved per rat; the castrates 5.8. The normal rats averaged 0.9 teeth totally destroyed per rat; the castrates 0.7. There is apparently a tendency for the normal rats to have somewhat higher average values per rat. However, these differences can be accounted for by the

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fact that only 1 castrate survived until the 19th month on the diet whereas 3 normal rats survived this period. It was in these oldest animals that the very high values for the numbers of cavities, of cusps involved, and of teeth totally destroyed were found. Consequently, it may be said that normal and castrate

TABLE I

Data on experimental caries in normal female rats

RAT NO.	DIED	AGE	MONTHS ON DIET	NO. OF CAVITIES	NO. CUSPS INVOLVED	NO. TEETH DESTROYED	CARIES FREE MOUTH
		months					
33	5/41	18	6	0	0	0	yes
25	10/41	23	8	4	10	0	no
40	10/41	23	8	2	4	0	no
29	11/41	24	9	2	6	0	no
37	1/42	26	11	0	0	0	yes
38	1/42	26	11	0	0	0	yes
38?	1/42	26	11	0	0	0	yes
22	2/42	27	12	1	3	0	no
31	2/42	27	12	6	23	3	no
31?	3/42	28	13	0	0	0	yes
34	4/42	29	14	2	7	1	no
40	5/42	30	15	1	1	0	no
23	6/42	31	16	4	8	1	no
37	8/42	33	18	4	4	0	no
22	9/42	34	19	7	26	5	no
30	9/42	34	19	6	15	2	no
80?	9/42	34	19	7	20	3	no

TABLE II

Data on experimental caries in castrate female rats

RAT NO.	DIED	AGE	MONTHS ON DIET	NO. OF CAVITIES	NO. CUSPS INVOLVED	NO. TEETH DESTROYED	CARIES FREE MOUTH
		months					
15	5/41	18	3	0	0	0	yes
17?	8/41	21	6	0	0	0	yes
15?	10/41	23	8	0	0	0	yes
10?	10/41	23	8	0	0	0	yes
12	10/41	23	8	0	0	0	yes
9	11/41	24	9	0	0	0	yes
7	11/41	24	9	0	0	0	yes
14	4/42	29	14	3	10	1	no
10	4/42	29	14	5	9	1	no
20	5/42	30	15	6	14	1	no
5	6/42	31	16	6	14	2	no
3	9/42	34	19	7	23	3	no

female rats in long continued exposure to the caries-producing diet do not differ in the incidence of carious lesions. Combining the data of Tables I and II it was found that little difference exists in the amount of caries in the various locations in the mouth. 32 upper teeth were affected and 42 lower; 39 teeth of the

maxillary and mandibular right jaws were affected; 35 teeth of the left jaws. The tendency for approximately equal caries susceptibility is various quadrants of the mouth has been repeatedly observed.

In *fig. 1* is shown (solid line) the rate at which the number of carious teeth appeared in all of these rats. The dotted line indicates the comparable values observed by many authors on the rate of caries attack in animals placed at weaning on the HWC diet. The numbers beside each point indicate the number of rats studied. The number of carious teeth after 6 months on the diet is strikingly different for young rats (3+) and for animals placed on the diet at the age of 1 year (0). For the latter animals 18 months on the dietary regime were required to produce as many carious teeth as are found in young rats after 6 months. There is evidence of two phases in the production of carious lesions in the teeth of the older rats. Up to 15 months the increment of carious teeth is small and the curve rises slowly. Thereafter the curve rises sharply so that after 19 months on the diet, the rats have about 7 carious teeth each. McClure

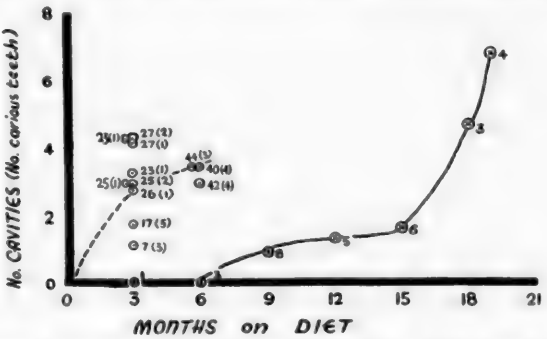

FIG. 1. *Increase of carious teeth with time on HWC diet.* Solid line is for rats placed on diet at age of one year. Dotted line is for weanling rats. Numbers beside each point refer to number of rats observed. Numbers in parentheses refer to the following authors, (1) McClure and Arnold (17); (2) McClure (18); (3) Cheyne (19); (4) Hodge and Finn (27); (5) Miller (11).

(28) noted that older rats placed on the coarse corn meal diet at the age of 200 days showed fewer carious teeth, fewer carious areas, and a lower caries score than younger rats, although the differences were not large. It is clear that year-old rats placed on the HWC diet develop carious teeth very slowly as compared with rats placed on the same diet at weaning.

In *fig. 2*, the average number of cusps involved for each rat are shown (solid line) to increase with increasing time on the diet. In comparison, some data on young rats are included (dotted line). The old rats dying after 6 months on the diet had no cuspal lesions whereas weanling rats had about 10 cusps involved per rat in the same length of time. An interesting feature is the tendency in older animals for the number of cusps involved to increase linearly with the time on the diet. The animals sacrificed after 19 months on the diet seem to have too high a number of cusps involved to fit the linear tendency. However, had these animals been allowed to live out their complete life span the aberration

might have disappeared. The linear tendency may have been only a coincidence; it deserves further study.

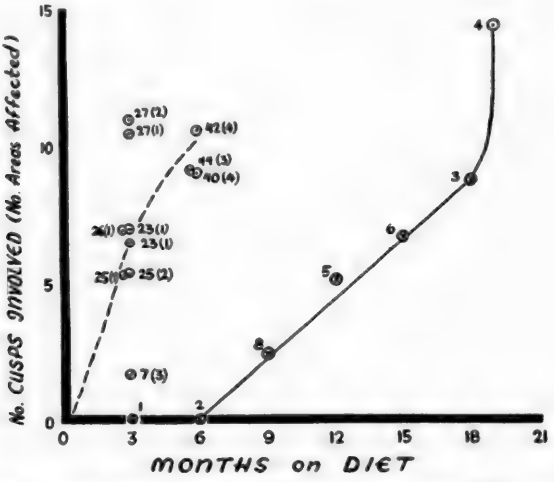

FIG. 2. *Number of cusps involved per rat with increasing time on HWC diet.* Significance of numbers is same as in *fig. 1.* Old rats, solid line—weanling rats, dotted line. Apparent tendency for linear increase in number of cusps involved in older rats is noteworthy.

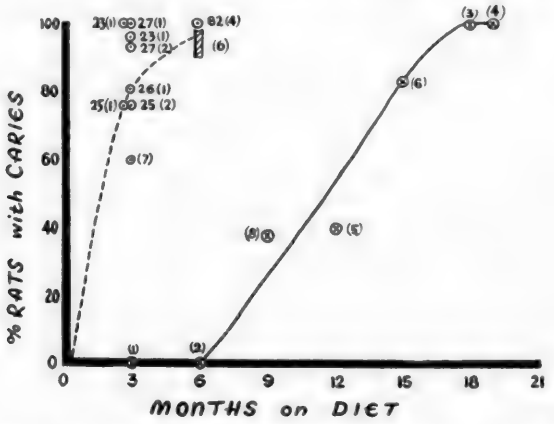

FIG. 3. *Percentage of rats with caries after various times of exposure to HWC diet.* Old rats, solid line,—weanling rats, dotted line. Significance of numbers is same as in *fig. 1* except that for young rats (6) refers to Hoppert, Weber, and Caniff (3), and (7) refers to Lilly (22).

In *fig. 3*, the percentage of the rats with caries is sbown (solid line) as a function of the time on the diet. For comparison, data on weanling rats on the same diet are given (dotted line). Whereas young rats develop caries in 90% to 100% of the colony in 6 months, none of the older rats had any caries in the same

period. In the ensuing year the older animals gradually showed an increasing incidence of caries and after 18 months on the diet all of them showed lesions.

It seems reasonably clear that rats aged 1 year when placed on a coarse corn diet are much more resistant to experimental caries than are weanling rats. The factors involved in lessening the susceptibility in older animals are not known. One can imagine that the processes of maturation described by histologists in human teeth may have some counterpart in rat teeth. What the maturation changes are and how they decrease the incidence of caries are not known. The well established decrease in the susceptibility of human teeth with age perhaps finds a counterpart in the data reported herewith for rats. Although this study is on only a few rats, it throws light on a phase of experimental rat caries which deserves additional investigation.

SUMMARY

1. 17 normal and 12 castrate female rats were placed at the age of 1 year on the coarse corn diet which was ingested up to 19 months thereafter.

2. No differences were observed between the caries incidence in the normal and in the castrate rats.

3. These rats developed caries much more slowly than do weanling rats placed on the same diet.

4. After 18 months on the diet, all the surviving rats had caries.

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TOOTH CONDITION, A FACTOR IN EXPERIMENTAL ISOTOPE ABSORPTION[1]

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INTRODUCTION

Experiments with radio-active phosphorus have not given uniform results. Differences of opinion still exist concerning a minimal degree of metabolism in the enamel and the rôle of the pulp in this respect. The reason may be that there is a general tendency to regard all teeth as being closely similar in structure, physical character and physiological activity.

The present paper shows evidence that the permeability of the teeth is variable, a quality closely related to physiological phenomena. This physical characteristic is affected by a number of conditions: 1. Tooth age, 2. Type of animal, 3. Degree of attrition, 4. Secondary dentin, 5. Degree of protective metamorphosis, 6. Degree of activity of the dental pulp. The first 4 conditions are known generally to histologists; they are mentioned only briefly so as to present all known factors which may affect the permeability of the teeth. The degree of protective metamorphosis (1) and activity of the dental pulp, however, have received no consideration and will be discussed in detail.

EXPERIMENTAL EVIDENCE

Permeability of the dental tissues is the most important variant in teeth. This physical quality differs, depending on the conditions named above. A most important factor is the post-formative age of teeth. Klein, Palmer and Kramer (2) have designated this as the post-eruptive tooth age; a better term, probably, would be post-formative tooth age. There is a difference between these terms. A tooth may be retained for years in its alveolus and yet in this position undergo the changes described below, just as if it really had taken its place in the mouth at the normal time. A tooth, therefore, having a delayed eruption would be designated as a younger tooth if the term "post-eruptive tooth age" were used than the one which had made its normal appearance in the mouth. The reason for this differentiation is that recently formed teeth, both in man and animals, are more permeable than those which have been formed for a long time (3). The type of the animal also has a bearing in this respect; the teeth of dogs, for instance, are more permeable than those of man (4). Wear or attrition of teeth sets up an irritation of the pulp by way of the dentin which causes the formation of secondary dentin; this further reduces their permeability (5).

[1] Read at The 21st General Meeting of the International Association for Dental Research, Chicago, March 14–15, 1943. (*J. D. Res.* **22:** 200, 1943.) Received for publication April 15, 1943.

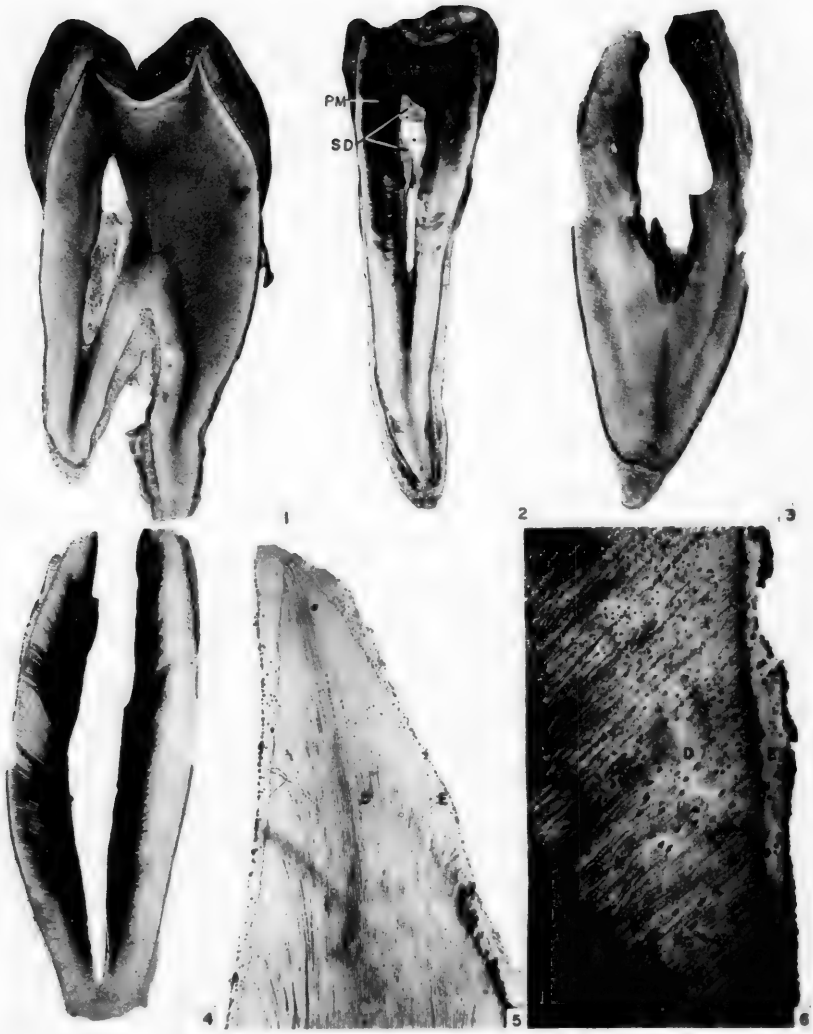

FIG. 1. Ground section of a young human tooth showing highly permeable dentin with exception of a very slight protective metamorphosis (PM).

FIG. 2. Ground section of old human tooth. Protective metamorphosis (PM) and secondary dentin (SD) formation make crown impermeable.

FIG. 3. Pulp of human tooth was removed 5 years previously. Tooth stained *in vivo* for 1 week. Permeability of dentin low as shown by slight dye penetration. (Ground section).

FIG. 4. Pulp of human tooth devitalized and stained *in vivo* 1 week later. Permeability of dentin high as shown by considerable dye penetration. (Ground section).

FIG. 5. Human tooth stained *in vivo* with silver nitrate 11 years after eruption. (Decalcified section.)

FIG. 6. Higher magnification of part of tooth shown in *fig. 5*. Minute particles of silver nitrate limited to dentinal tubules (left upper). Elements of enamel matrix completely stained by dye originating from pulp (secondary staining). See also *fig. 5* in Lefkowitz, *J. D. Res.* **22:** 290, 1943 (this issue).

All of these agencies—tooth age, type of animal, attrition and formation of secondary dentin—must be taken into account in interpreting the degree of absorption of isotopes used for the purpose of defining whether or not slight metabolic processes take place in dentin and enamel. The remaining two factors mentioned in the introduction will be discussed in detail.

Protective metamorphosis has been demonstrated by histological observations (6, 7) and in *in vivo* experiments (4). Beust (5) made extensive observations on this subject defining the varying permeability of dentin. It has been shown (7, 8) that the change in the contents of the dentinal tubules, as a result of a physiological ageing process is hastened by peripheral irritation (attrition, caries) causing many of them to become blocked. A comparison of *figs. 1 and 2* will show the difference between a "young" and an "old" tooth. The reduction in permeability in "old" teeth has been termed "protective metamorphosis" because it protects the pulp from external irritation and reduces the rate of caries penetration. As a result of the blocking of the tubules, the dental lymph can no longer penetrate such areas, thus eliminating certain portions of the dentin from pulpal connection.

The degree of vitality of the dental pulp. Some of the functions of the dental pulp are to furnish nutrition to the dentin (9) and form secondary dentin as a result of the external irritation of attrition and dental caries thus protecting it from exposure. Logically, when the pulp dies, these functions cease. Observations (1–4), however, present evidence that the death of the pulp not only causes a cessation of these activities, but it does more. While the pulp is vital, the flow of dental lymph (tissue fluid), most active in young teeth, occurs principally through organized channels; i.e., the dentinal tubules, enamel tufts, spindles, lamellae, and rod sheaths. When pulp necrosis occurs in a recently erupted tooth, the permeability of the dentin and enamel is vastly increased temporarily, so that experimental dyes diffuse through these tissues profusely, irrespective of the usual channels of communication. The tooth of a dog, for instance, takes up the dye in the same manner that a sponge takes up water (4).

Some time after pulp removal the permeability of the dentin is again reduced so that it becomes less than that observed in vital teeth. *Fig. 3* is a section of a canine of a 57-year old person which had been pulpless for more than 5 years. The filling was removed, and the root canal enlarged after which gentian violet was inserted. Only slight staining is noted after 1 week's time. *Fig. 4* was a vital canine of the same patient. The pulp was extirpated and root canal filled. One week later gentian violet was inserted subsequent to the removal of the canal filling. After the lapse of 1 week considerable stain penetration was noted.

DISCUSSION

The absorption of slight amounts of isotopes by the dentin and enamel suggests that a minimal degree of metabolism is present in these structures [McCauley (10)]. Histological observations (6, 7) and vital staining methods (4) showed the presence of permeable channels in the dentin and enamel proving their connection with the dental pulp. In this respect, there is a certain advantage of the *in vivo* staining method over the use of the isotopes, inasmuch as the

histological channels are clearly defined by the dyes used in the vital staining technique. Isotopes can be traced only in macroscopically different areas, such as in the outer or inner layers of the enamel and in the outer, middle or inner portions of the dentin. The vital staining method, on the other hand, shows the histological structures that carry the dissolved dyes. This method appears to have some merit not possessed by the technique of isotope injections, inasmuch as the former demonstrates the pathways of the dental lymph. *Figs. 5 and 6* demonstrate this localization. *Fig. 5* is a section of a human second incisor of a patient aged 17 which shows silver nitrate completely permeating the crown. The dye was placed in an experimental cavity at the cervical portion of the tooth. The fact that this tooth was comparatively young, having a post-formative age of 11 years explains the deep penetration of the dye through dentin and enamel. *Fig. 6* is a higher magnification of the labial portion of the same tooth seen in *fig. 5*. Here it is noted that the very fine granules of silver nitrate (left upper corner) are localized in the dentinal tubules. The entire thickness of the enamel including its surface has been stained.

Further, the vital staining method throws light on the reason why certain areas of the dentin and enamel are cut off from the dental lymph (tissue fluid). Examination under the microscope indicates that some groups of dentinal tubules are blocked by protective metamorphosis and that this blocking increases as a result of the physiological ageing of the tooth. In *fig. 2* the entire crown has become impermeable.

All in all, the placing of a dye in individual teeth seems to throw more light on the particular channels through which physiological activity of the pulp affects the dentin and enamel than does the injection of radio-active phosphorus.

It has been shown here and in another work (1) that the removal of pulps from recently formed teeth causes a considerable increase in permeability (4). The observation of Osterhout (11) suggests that dentin and enamel react in a manner similar to vital tissues. He states that: "It is well known that death is accompanied by an increase of permeability. Thus a slice of red beet kept in water will live for a long time without giving off pigment; but as soon as it is killed, the color begins to escape from the cells. . . . It is a matter of common observation that cells may resist the penetration of certain dyes as long as they are alive, but absorb them readily as soon as they are killed."

The knowledge that the removal of the pulp allows an abnormal entrance of blood plasma into the tooth from the periodontal membrane shows that such experimental observations are not a reliable index of the metabolism of the calcified dental tissues.

One more point must be considered concerning the possible source of radio-active phosphorus found in enamel. Wassermann, Blayney and Groetzinger (12) excluded the saliva by placing caps on certain teeth. There is no doubt that such a barrier prevents the deposition of the radio-active phosphorus on the enamel, but it may have a further effect of reducing the osmotic flow of the dental lymph (tissue fluid). It is possible, therefore, that such caps account for a reduction in the amount of radio-active phosphorus in the enamel. This subject is being investigated.

The above observations show that the experimental use of radio-active phosphorus employed to define whether or not metabolic activity exists in dentin and enamel requires a preliminary histological examination of axial ground sections of experimental teeth, so as to ascertain their degree of permeability at the time of the experiment. Teeth, such as rat molars, showing extensive attrition and secondary dentin formation can be expected to receive only a minimal amount of radio-active phosphorus from the dental pulp. Hence the judgment of whether or not metabolic activity exists in enamel and dentin should be modified as applying either to young, recently formed teeth or those that are highly matured.

Confusion results if the above variables are not taken into consideration inasmuch as the pulp is regarded erroneously as being unimportant in effecting metabolic changes in the tooth.

SUMMARY

The amount of radio-active phosphorus defined in dental tissues as a result of experiments designed to determine the degree of their metabolic activity is dependent largely upon the age of the tooth, type of the animal, secondary dentin formation, protective metamorphosis, degree of attrition and pulp vitality. All of these factors affect the permeability of teeth, a quality closely related to physiological phenomena. If protective metamorphosis has occurred due to ageing, attrition, and secondary dentin formation, permeability of the dentin is reduced greatly; hence, very little radio-active phosphorus can pass from the pulp into the dentin and enamel. The teeth of animals used for purposes of defining metabolic activity should be examined as to their permeability because this factor will affect the results of the experiment.

The degree of pulp activity also plays a rôle in the degree of metabolic activity in dentin and enamel. Pulp extirpation leads to a vast temporary increase in permeability in recently erupted teeth, causing the absorption of radio-active phosphorus from channels other than the dental pulp. Thus a confusion results, suggesting that the dental pulp is unimportant in effecting metabolic changes in the tooth.

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FURTHER OBSERVATIONS OF DENTAL LYMPH IN THE DENTIN[1]

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The results of the early investigations of staining teeth *in vivo* were examined in ground sections (1, 2, 3). The dye, placed in a cavity in the dentin, was permitted to remain in situ for periods varying from 5 to 28 days before the specimens were taken. The period of staining was based on the concept that the rate of diffusion of dental lymph was slow. Subsequent examination of the ground sections showed the stains confined to the area of the cut tubules and a strand of intact tubules adjacent to the stain cavity. It was further observed (3) in ground section examination of vital staining that some of the dye, placed in the stain cavity in the dentin, stained areas of uncut tubules close to the pulp chamber.

Attempts to study the results of vital staining of dentin in decalcified sections proved successful with the use of silver dyes (4). At the same time, the staining period was reduced to hours instead of days. The reduction of time in these experiments stimulated the following investigation of the rate of flow of dental lymph (tissue fluid)[2]. This report is concerned principally with the outward diffusion, i.e., from the pulp to the periphery of the dentin.

THE RATE OF PERIPHERAL FLOW OF DENTAL LYMPH

The teeth of a dog were stained *in vivo* with argyrol for periods varying from 13 to 58 minutes, at intervals of 3 minutes. A small cavity approximately ½ mm. in diameter was prepared at the cervix into the pulp. The dye was inserted therein and the cavity sealed. Thirteen teeth stained with argyrol were examined and the results reported in this paper.

RESULTS

In all cases the dye permeated the pulp. The greatest intensity was in the coronal area diminishing toward the apex where no staining was observed. In the dentin the greatest concentration of particles was in the predentin and pulpal area of dentin diminishing toward the periphery. The silver was distributed through the dentinal tubules and followed their curvatures.

The flow of dental lymph (tissue fluid), demonstrated by the distribution of the dye, appears to be directed toward the crown and cervical half of the root. The intensity of the stain varied with the length of time of the experiment. Similarly, the distribution of the dye through the tubules was in accordance with the longer period of staining.

[1] Presented at the 21st General Meeting of the International Association for Dental Research, March 14, 1943. (*J. D. Res.* **22**: 208, 1943.) Received for publication April 13, 1943.

[2] The term "tissue fluid" has been inserted, by the editor, after "dental lymph" at each place in this paper in order to call attention to the fact that this fluid, described by Bödecker as a product of the odontoblasts, is not lymph in the sense of that it is not within the lymphatics and does not contain lymphocytes.

The minute dye particles could not be photographed at a sufficiently low magnification to show the entire specimen. Hence, a diagram was prepared showing these results (*fig. 1*). It may be observed that 13 minutes after insertion of the dye, the penetration was sparse and extended about half the length of the tubules. The pattern of distribution is suggested, corresponding to the crown and cervical half of the root. In 17 minutes, although slightly denser, it had penetrated to the periphery of the tubules in the cervical area. In 28 minutes the staining was intense and extended to the terminal ends in all the stained tubules. The 38-minute diagram shows a more intense stain and slightly wider distribution.

The pattern of distribution followed the same form throughout all the specimens. The diffusion of dental lymph (tissue fluid) is directed toward the crown. The stained area increased in size up to the 38-minute specimen and remained comparatively static thereafter.

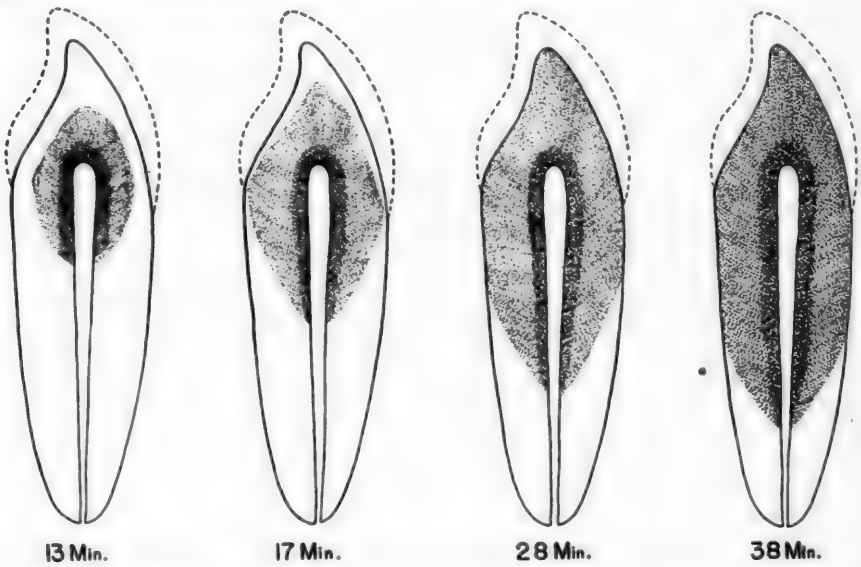

FIG. 1. Diagram showing rate of flow of dental lymph (tissue fluid) in teeth of young dog

Fig. 2 is a photomicrograph of the crown of a specimen stained for 13 minutes. The illustration was used as a basis for the diagram for the same staining period. The distribution of the dye may be observed having passed half the length of the tubules. The profuse stain in the circumpulpar dentin may be better seen in *fig. 3* under a magnification of 175 times.

The uniform distribution of the dye to the periphery of the tubules may be observed in *fig. 4*. This is the 28-minute specimen shown in *fig. 1*. The intensity of the stain cannot be properly evaluated under a 50 × magnification. The particles visible in this illustration and *fig. 2* have been described as large particles, i.e., considerably larger than the tubules. The smaller and more numerous particles, which are localized particularly within the dentinal tubules, cannot be observed under this magnification.

TWO WAY FLOW OF DENTAL LYMPH IN DENTIN

The two-way diffusion of dental lymph may be observed by placing a dry dye on a stain pocket prepared into the dentin. For purposes of description 2 results in staining can be identified. One may be observed in the tubules cut in preparation of the stain cavity. Here the dye passed inward to the pulp and outward toward the periphery. The cut tubules stain very intensely and the particles are comparatively large. After the dye has reached the pulp, the former passes outward again. Here it is less intense and the particles are both large and small. This has been termed secondary staining (3).

The distribution of a dye in 2 directions has been described in previous reports (3, 4, 5). The original observation was made on ground sections in which the cut tubules were examined. Nevertheless, the dye diffused from the stain pocket to the periphery of the dentin and into the deep layers of the enamel in the peripheral flow. In the opposite direction the dye diffused to the pulp. Subsequently, decalcified sections suggested the mechanism by which this was possible. Evidence was presented that the peripheral flow occurred through Tomes' fibrils and the pulpal flow through the perifibrillar space (4).

These observations were made, in the main, on the teeth of dogs. Human material was confined to teeth of older individuals because of the difficulty in securing sound teeth of young persons. The histologic picture in the teeth of young human beings is typical of that found in dogs. *Fig. 5* is from a section of a second incisor of a 17-year old individual. A stain pocket was prepared in the dentin and argyrol sealed therein for 6 hours. The result graphically describes the entire picture of dental lymph diffusion. The stain pocket (SP) was prepared in the dentin. The cut tubules (CT) were intensely stained. No explanation of this unusual reaction is offered at this time. The argyrol, as can be observed, passed to the periphery of the dentin and into the organic matrix of the enamel. The pulpal flow may be observed by the intense stain from the stain pocket to the pulp. The dye, distributed throughout the coronal and cervical half of the pulp, was redistributed throughout the crown and cervical dentin (SS). The use of silver dyes in decalcified sections demonstrates a wider distribution of the secondary stain than previously observed (3). *Fig. 6* is a higher magnification of the area of secondary staining showing the intensity of the stain in this area, which is apical to the cervix of the same specimen shown in *fig. 5*. The stain particles may be observed in the predentin. The dye may also be observed in the organic matrix of the enamel. It is interesting to note that the enamel overlying the cut tubules was stained as well as the enamel overlying the area of secondary staining. (Higher magnification of the stained enamel matrix may be observed in an article by Bodecker (6)).

Thus it may be observed that a dye placed in a stain pocket in the dentin will diffuse both peripherally and pulpally in the area of the cut tubules. The dye which entered the pulp through the tubules was redistributed peripherally similar to the pattern of distribution as described in *fig. 1*. Furthermore, the dye also penetrates the enamel in both the area of the cut tubules and the area of secondary staining. This specimen is offered to corroborate the observations made on ground sections and animal material.

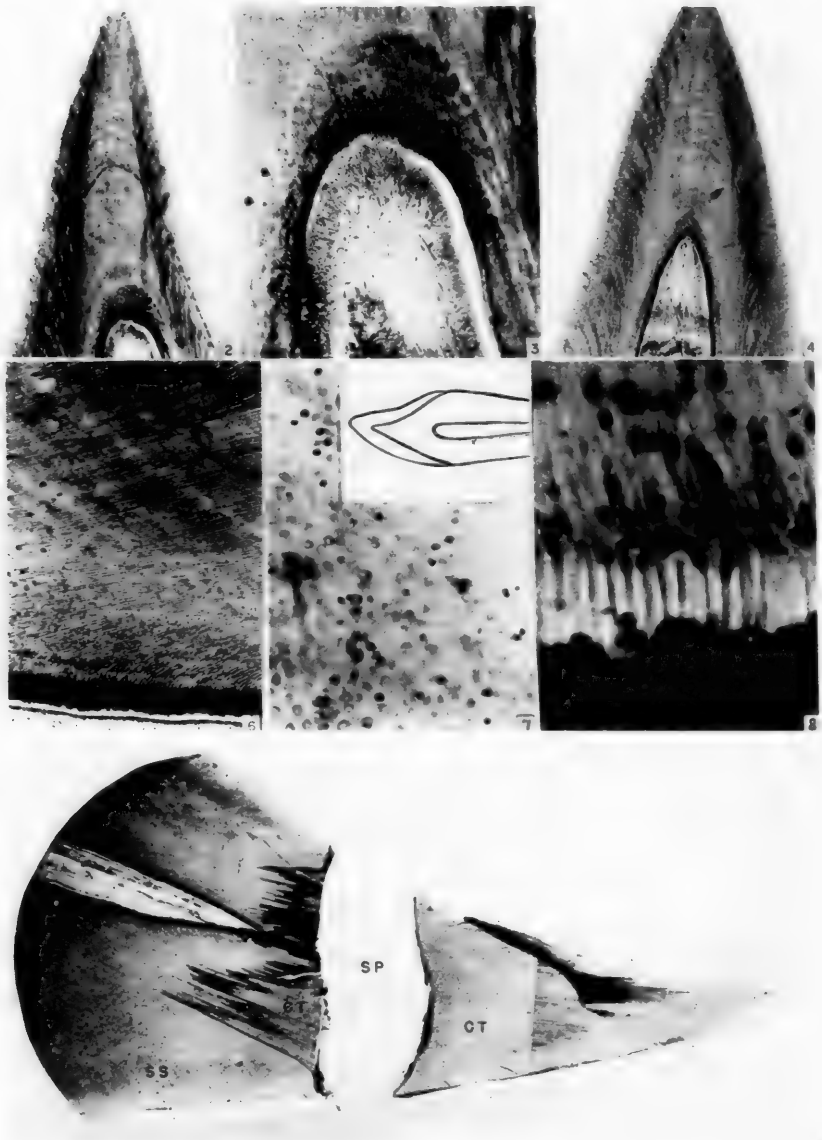

FIG. 2. Photomicrograph of tooth of dog stained 13 minutes with argyrol placed in pulp. Dye penetrated approximately half length of tubules. 55 ×.

FIG. 3. Higher magnification of same specimen as *fig. 2* showing profuse staining of pulp, predentin and dentin. 175 ×.

FIG. 4. Photomicrograph of tooth of dog stained 28 minutes with argyrol placed in pulp. Dye particles have penetrated entire length of tubules. 50 ×.

FIG. 5. Photomicrograph of human second incisor—age 17 years. Argyrol deposited in stain pocket (SP) for 6 hours. Cut tubules (CT) stained intensely to pulp and periphery

The short period of staining described in the first half afforded a splendid opportunity to determine the diffusional channel of the peripheral flow. *Fig. 7* is a photomicrograph of a section cut through the predentin of a dog's tooth which had been stained *in vivo* for 17 minutes described in *fig. 1*. The dye had not penetrated the entire length of the tubules in this area. Only fibrillar staining may be observed in this section which is conclusive evidence that the peripheral flow occurs through Tomes' fibrils as described by Bodecker (7). This was also observed in the human specimens in longitudinal sections of Tomes' fibrils which were isolated by shrinkage of the pulp. The stain particles were observed in the isolated fibrils extending into the predentin.

An examination of the predentin and frequently of the dentin shows that Tomes' fibrils stain deeper with hematoxylin than the basis substance. This stain is considered an indicator of the degree of calcium present before decalcification of the specimen. The failure of uncalcified basis substance in predentin or interglobular dentin to take hematoxylin establishes the value of this stain to demonstrate the degree of calcification. Meyer (8) uses this method to describe linear and globular calcification of dentin. Orban (9) uses it to describe the quality of calcification. *Fig. 8* is a photomicrograph of a section of predentin, from another second incisor, of a 17-year old individual showing Tomes' fibrils deeply stained with hematoxylin.

DISCUSSION

The rate of diffusion of dental lymph (tissue fluid) from the odontoblast to the periphery may be measured by the extent and intensity of the dye diffusion. These results are not to be accepted literally because of many variables. All the specimens came from the same animal but the teeth varied in post-eruptive age. The animal could be described as young. The diffusion of dental lymph (tissue fluid) is greater in the teeth of dogs than in human teeth. It also diminishes with age in human teeth.

The amount of argyrol inserted into the pulp was not measured. Whether or not the quantity of dye used would materially affect the result cannot be stated at this time.

The information derived from this investigation cannot be transferred to human material. It does, however, demonstrate that the diffusion of dental lymph may be measured in minutes and that the previous concept of the rate of flow of dental lymph (tissue fluid) measured in days or hours is erroneous.

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of dentin. Secondary stain (SS) in cervical area. Note stained enamel matrix in both area of cut tubules and secondary stain. 17 ×.

FIG. 6. Area of secondary stain taken from cervical dentin of *fig. 5*. Note profusion of large and small stain particles. Some of precipitate also visible in predentin. 125 ×.

FIG. 7. Transverse section through predentin of root of dog tooth stained with argyrol in pulp 17 minutes. Dye had not penetrated entire length of tubules. Only peripheral flow possible. Stain particles may be observed in Tomes' fibrils. No perifibrillar staining. 1500 ×.

FIG. 8. Predentin of human second incisor, age 17. Note Tomes' fibrils stained with hematoxylin. 1500 ×.

The pattern of distribution suggests, as observed in human and animal specimens, that the flow of dental lymph (tissue fluid) is more profuse in the coronal portion of the tooth and diminishes toward the apex. This may be the result of having inserted the dye at the cervix. The diffusion is dependent on the odontoblasts.

This area of root dentin also demonstrates a failure to regularly undergo protective metamorphosis. The correlation of these observations suggest that the failure to observe the same stain reaction may be attributed to the odontoblastic activity and not the original location of the stain in the pulp.

The presence of a two-way flow of dental lymph (tissue fluid) in the dentin and its diffusion into the enamel is demonstrated in young human material. The significance of this finding establishes the need for research into the physiology of the calcified dental tissues. The suggestion that dental lymph (tissue fluid) contains calcium may lead to investigation to establish a function for the odontoblast.

The deep staining of Tomes' fibrils with hematoxylin is best observed in the predentin where the basis substance takes eosin. The observation suggests a high calcium content in dental lymph (tissue fluid) and that calcification of the dentin may occur through this activity which originates in the odontoblast.

CONCLUSIONS

The rate of flow of dental lymph (tissue fluid) from the pulp to the periphery of the dentin may be defined in minutes rather than hours. It follows a pattern of distribution which is concentrated in the crown and cervical half of the root. A two-way flow of dental lymph (tissue fluid) in the dentin is demonstrated in young human material. Evidence that the peripheral flow occurs through Tomes' fibrils is further substantiated. A suggestion that dental lymph (tissue fluid) contains calcium is made on the basis of its staining reactions with hematoxylin.

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INITIAL OXIDATION-REDUCTION POTENTIALS OF SALIVA[1]

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Oxidation-reduction studies of saliva have been neglected, possibly due to the rapid drift toward a negative potential under *in vitro* conditions at room temperatures. To the writer's knowledge only 3 references appear in the literature. Pincus (1) first reported the marked reducing properties of saliva as demonstrated by the reduction of methylene blue and dichlorphenol-indophenol to the colorless form within a few seconds. Appleton (2) indicated the need for studying the oxidizing and reducing properties of saliva. He observed that the ability of saliva to reduce methylene blue varies with the individual and may even be absent. Aerobic bacterial flora of the mouth may partially account for the reducing power of saliva. It should be pointed out that bacterial dehydrogenases in the presence of suitable substrates could easily account for a low E_h and especially the reduction of methylene blue. Wessinger (3) made a few tests with an electrometer by placing saliva in a sample cup and testing *in vitro* at room temperatures, but found that the potential drifted rapidly to the negative scale, even under paraffin oil. He found no correlation between the potentials and pH and concluded that no accurate data on oxidation-reduction activities of saliva were obtainable.

The general field of oxidation-reduction is growing rapidly in biology, but the drifts in biological systems have caused great difficulties in obtaining consistent results. Good reviews of the literature are found in Elvehjem and Wilson (4) and Michaelis (5). The purpose of this paper is to report the temporary oxidation-reduction activities of saliva immediately after spitting, and to determine whether or not consistency of data is obtainable. Repeated oxidation-reduction tests were made in this laboratory while studying the hydrogen-ion concentration of the same sample of saliva (6).

METHODS

The procedure was that used by Eisenbrandt (6) for determining the pH of saliva. A pH-E_h meter was utilized with a gold-plated platinum electrode which was substituted for the glass electrode used in determining the pH of saliva. This instrument was identical to the one used by Wessinger (3). Unstimulated saliva was collected within the closed mouth until about 5 ml. had accumulated; then it was spit into the sample cup and tested immediately. Only a fraction of a minute was required to switch electrodes, adjust the instrument and obtain the potential readings at temperatures averaging 28°C. Consequently both pH and E_h readings were obtained within 1½ minutes after filling the sample cup with saliva. The tests were entirely *in vitro*.

[1] Received for publication July 9, 1943.

Preliminary tests indicated that little change occurred in the readings during the short interval required for testing; also, the potentials probably were nearly equivalent to that of resting saliva within the mouth. When electrodes were placed directly into the mouth the salivary glands were stimulated somewhat, therefore, the method used in the tests was believed more desirable.

Usually after 3 to 5 minutes the oxidation-reduction potentials of saliva *in vitro* began to drop irregularly whether exposed to air or sealed under paraffin oil. Because of the short time interval required for the determinations, a closed or sealed system was deemed unnecessary. It is emphasized that the E_h values obtained were initial ones read at the outset of the gradual drift of the potentials and were not the terminal points. The saliva of each subject was tested 5 times during the day for 10 to 12 days during October and November, 1942. The data were studied statistically using the methods advocated by Snedecor (7); the tables present the results in a summarized form. The oxidation-reduction potentials (E_h) were expressed as millivolts.

EXPERIMENTAL DATA

The initial oxidation-reduction potentials (E_h) of saliva of 5 subjects were tested 5 times daily; a total of 252 readings were accumulated. The mean E_h for the group was $+261 \mp 1.88$ mv. (the last number is the standard error of the mean and not the standard deviation). In Table I are the mean E_h for the group and for each individual. Approximately 50 readings were obtained for each subject. By determining the total mean ratio between the group mean and each subject, it was found that no individual was significantly different, although C. approached significance by having a mean of +252 mv. Therefore, it may be said that the group mean was fairly representative of all individuals and that no one deviated greatly from the mean.

An analysis of variance (Table I) approached the problem from the viewpoint of how the subjects varied among themselves. The ratio between the mean square of the 2 sources of variation indicated that the subjects were not significantly at variance with one another. Therefore, the oxidation-reduction potential of saliva of each person was much like that of the others and did not show characteristics peculiar to any one subject. (Compare this with Tables I and II in the writer's earlier paper on the pH of saliva of these same individuals.)

The data were considered next from the standpoint of the bi-hourly intervals of 9 A.M., 11 A.M., 1 P.M., 3 P.M. and 5 P.M. (Table II). The mean E_h for each period was analyzed to find if it was significantly different from the total group mean of +261 mv. No bi-hourly period was significantly different; however, the greatest differences were at 9 A.M. and 5 P.M. and no difference was indicated at 1 P.M. It is to be noted that there was a general downward trend in E_h as the day progressed. Based upon the coefficient of correlation the E_h of saliva for the bi-hourly periods was significantly related, inversely, to the pH previously reported for the same periods. The coefficient of correlation was −0.28 which led one to believe the E_h and pH were not related. However, when this number was tested for significance the t value was 0.160 and the value required for 1% significance was 0.163 which left little doubt of an inverse relation-

ship (*fig. 1*). Each individual graph was quite similar to the mean presented here; no one was markedly different. The inverse relationship between pH and E_h is marked especially during the early morning and the late afternoon.

TABLE I

Initial oxidation-reduction potentials of saliva of 5 individuals

	S	C	P	E	H	GROUP
No. readings	46	46	50	50	60	252
Mean E_h (mv.)	+263	+252	+254	+267	+265	+261
Std. deviation	±27.8	±27.7	±26.8	±25.5	±33.7	±29.7
Std. error of mean	∓4.10	∓4.08	∓3.79	∓3.61	∓4.35	∓1.88
Total mean ratio	0.49	2.20	1.85	1.66	0.92	
1% Significance	2.69	2.69	2.68	2.68	2.66	

Analysis of variance

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARES
Total	251	224.492	
Between subjects	4	7.830	1.9575
Between readings of each subject	247	216.662	0.877

F = 1.9575/0.877 = 2.23.....not significant
1% Significance = 3.40

TABLE II

Bi-hourly variations in initial oxidation-reduction potentials of saliva of 5 individuals

	9 A.M.	11 A.M.	1 P.M.	3 P.M.	5 P.M.	GROUP
No. readings	53	51	50	50	48	252
Mean E_h (mv.)	+270	+256	+261	+260	+250	+261
Std. deviation	±37.6	±28.0	±27.8	±26.7	±25.9	±29.7
Std. error of mean	∓5.17	∓3.90	∓3.93	∓3.78	∓3.74	∓1.88
Total mean ratio	1.74	1.28	0.0	0.27	1.60	
1% Significance	2.67	2.68	2.68	2.68	2.69	

Analysis of variance

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARES
Total	251	224.492	
Between bi-hourly periods	4	7.6057	1.9014
Between readings in each period	247	216.8863	0.8781

F = 1.9014/0.8781 = 2.17.....not significant
1% Significance = 3.40

An analysis of variance of the group saliva for the 5 bi-hourly periods showed no variations, therefore, it can not be said that the E_h of saliva varied to any marked extent during the day. *Fig. 2* summarizes the data in Table II in graphic

form. The E_h became less at 11 than at 9 o'clock in each case, and between 11 A.M. and 1 P.M. all potentials increased except E; however, his increased between 1 and 3 P.M. After 1 P.M. considerable variation occurred among the subjects.

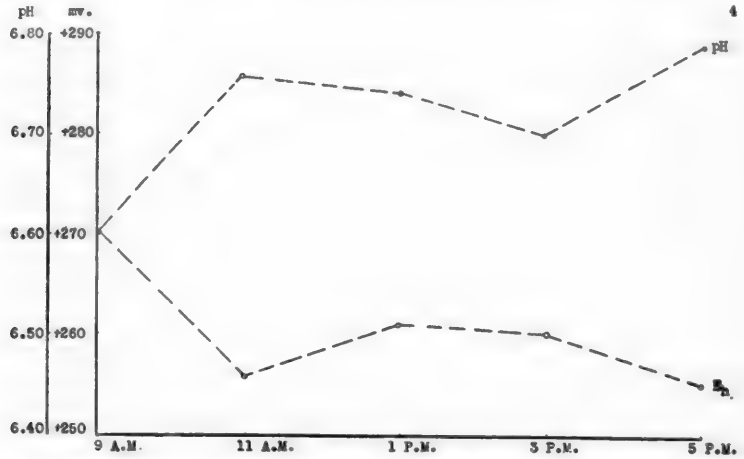

FIG. 1. Relationship between the mean pH and mean Eh of saliva of 5 subjects

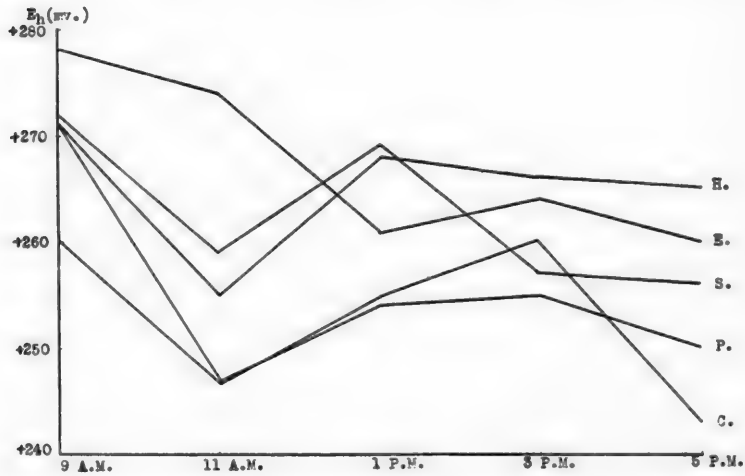

FIG. 2. Average bi-hourly variations in initial Eh of saliva for each subject

The remaining data (summarized in Table III) dealt with bi-hourly comparisons of each subject's saliva, individual differences from each bi-hourly mean and from the total mean of each subject. In each test there was no significant difference, indicating no particular individual characteristics. An analysis of

TABLE III

Mean initial oxidation-reduction potentials of saliva of each individual at bi-hourly intervals. Analysis of variance; individual variations for each period and for day. Also, individual differences from bi-hourly means and from daily mean

	9 A.M.	11 A.M	1 P.M.	3 P.M.	5. P.M.	DAILY	INDIV. ANALY. VAR. DURING DAY	
							F value	F required for 1% sign.
(S.) Mean E_h (mv.)	+272	+259	+269	+257	+256	+263	1.43	13.74
Std. error M.	∓9.4	∓1.0	∓11.8	∓9.1	∓4.0	∓4.1		
Ratio with daily mean	0.96	0.40	0.51	0.66	1.73			
Ratio with bi-hourly mean	0.21	0.30	0.68	0.33	0.23			
1% significance	3.17	3.36	3.36	3.25	3.71			
(C.) Mean E_h (mv.)	+260	+247	+255	+260	+243	+252	1.47	13.74
Std. error M.	∓13.3	∓8.2	∓9.5	±7.1	∓3.9	∓4.1		
Ratio with daily mean	0.60	0.61	0.32	1.11	2.31			
Ratio with bi-hourly mean	0.75	1.10	0.63	0.0	3.08			
1% significance	3.25	3.25	3.36	3.50	3.56			
(P.) Mean E_h (mv.)	+271	+247	+254	+225	+250	+254	1.20	3.77
Std. error M.	∓11.1	∓6.3	∓5.4	∓6.0	∓10.9	∓3.8		
Ratio with daily mean	1.54	1.11	0.0	0.17	0.04			
Ratio with bi-hourly mean	0.09	1.43	1.30	0.83	0.46			
1% significance	3.25	3.25	3.25	3.25	3.25			
(E.) Mean E_h (mv.)	+278	+274	+261	+264	+260	+267	1.32	13.72
Std. error M.	∓13.6	∓10.2	∓8.8	∓6.7	∓5.1	∓3.6		
Ratio with daily mean	0.81	0.69	0.68	0.45	1.38			
Ratio with bi-hourly mean	0.59	1.77	0.0	0.60	0.98			
1% significance	3.25	3.25	3.25	3.25	3.25			
(H.) Mean E_h (mv.)	+271	+255	+268	+266	+265	+265	3.18	13.68
Std. error M.	∓11.4	∓9.2	∓8.5	∓11.6	∓10.2	∓4.3		
Ratio with daily mean	0.53	1.09	0.35	0.09	0.0			
Ratio with bi-hourly mean	0.71	0.11	0.82	0.52	0.98			
1% significance	3.11	3.11	3.11	3.11	3.11			
Bi-hourly Mean	+270	+256	+261	+260	+255			
Bi-hourly analy. variance; F value	3.24	1.67	3.44	3.60	1.09			
F required for 1% significance	13.68	3.76	3.73	13.74	3.79			

variance was determined for each subject for the bi-hourly intervals, and also among the subjects at each period. The data were not significant. This in turn is interpreted to mean that the contents in Table III were significant in showing that the variance of the subjects and of the bi-hourly periods was not under conditions herein presented. Also, the subjects were nearly identical to the group mean and they did not vary to any marked extent during the day.

DISCUSSION

The fact that nearly all the data presented in this experiment showed no subject significantly different from the average of the group, and no significant variation among the subjects, suggests that the average E_h of +261 mv. represented an initial oxidation-reduction potential lasting approximately 2 or 3 minutes which was characteristic of all the saliva immediately following spitting by the subjects. This average was not the end point. Preliminary tests to determine the end point based upon 24 hour samples gave variable results ranging from +50 mv. to −100 mv. Often these results became constant after two or three hours.

The above average is given added value by the lack of significance of bi-hourly variations of the group during the day, by each individual not varying significantly at any hour from his total mean and from the mean for each period during the day. Therefore, it seems probable that these particular subjects had potentials which were essentially alike. The pH and E_h of saliva of the 5 subjects are in contrast, because the variations in pH were sufficiently great to give statistically significant data (6), and those of E_h were not. It is emphasized that these data apply only to the particular experimental conditions employed. Additional work now in progress suggests changing environmental conditions may introduce added complicating factors not readily interpreted.

The present data are not in complete agreement with Appleton's (2) observation that the reducing power of saliva varies with the individual. He did observe some exceptions to the reducing ability of saliva. Although individuals did vary in the present study, the variations observed were not statistically significant. Pincus (1) believed that the reduction was not entirely due to bacteria because heating to 55°C. for 5 minutes definitely lessened the reaction as did enzyme poisons and serum. Probably foreign organisms, enzymes, food, desquamated epithelium of the oral mucosa and saliva itself play a part in the E_h of normal saliva. The determination of the part played by these factors should be given future consideration.

SUMMARY

Initial oxidation-reduction potentials of unstimulated saliva of 5 subjects immediately following spitting showed no significant differences from the mean E_h of +261 mv. Approximately 50 tests were made upon each subject, and the total number of tests was 252. The subjects did not vary significantly from one another. At bi-hourly intervals during the day the E_h for the group became less as the day progressed, the greatest change occurred in the morning between 9 and 11 o'clock. However, the changes were not sufficiently great to be significant.

Each subject followed the mean for each bi-hourly period so closely that no one deviated significantly, and neither did the subjects vary from one another at any one period to be of significance.

Therefore, importance is placed upon the mean E_h of saliva of these subjects rather than upon any individual or bi-hourly characteristics.

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CLINICAL AND BACTERIOLOGICAL STUDY OF 340 PULP THERAPY CASES[1]

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Although x-rays, thermal and electrical vitality tests are commonly employed in diagnostic procedures associated with pulp canal therapy, reports correlating the results of these diagnostic procedures (1, 2) or comparing them with bacteriological data (3, 4, 5, 6, 7) are extremely limited. The present study is an attempt to evaluate the clinical and laboratory findings in selected cases of root canal therapy.

METHODS AND MATERIALS

Three hundred and forty root canal cases were included in this investigation. The ages of the patients ranged from 11 to 49 years, all but 24 of the individuals being under 35 years of age. Table I shows the distribution of the cases among the various teeth. Maxillary teeth constituted 270 of those treated, the remaining 70 being mandibular teeth. In the maxilla, chiefly anterior and premolar teeth received therapy, only 19 maxillary molars being included. In the mandible most teeth treated were premolars and molars, only 5 lower anteriors being represented.

Dental caries exposed 286 (84.1%) of the pulps, accident or trauma accounted for 40 (11.7%), operative procedures involving pulpal horns were responsible for 12 cases (3.5%) and 2 remaining cases were previously treated pulp canals reopened to correct ineffective therapy.

The teeth selected for root canal therapy were radiographed and tested for vitality before the beginning of operative work. All radiographs were taken with a standardized technic on intermediate Eastman films using 10 M.A. current and 45,000 volts. Although the limitations of x-ray diagnosis were recognized, an attempt was made to divide the cases into 2 main groups, x-ray positive and x-ray negative. Cases in the x-ray positive group showed a definite break in the lamina dura with loss of normal bone architecture around the apices. The group was further divided into cystic and non-cystic lesions after root resection and subsequent histological diagnosis. X-ray negative cases possessed intact periapical areas.

The vitality of the pulp was examined with the Burton high frequency pulp tester operating on 110 volt A.C. circuit and by thermal tests. The electrode of the pulp tester was disc shaped with a concave surface. A tooth without caries or restorations was isolated and tested by applying the electrode to the labial surface and observing the point upon the scale at which a response was elicited.

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The tooth in question was examined in the same manner and its reaction point noted and a gross indication of the vitality or lack of vitality of the pulp was obtained by comparison. Reaction to heat was tested by means of gutta percha heated until soft or by a burnisher heated until red. The heat agent was then touched to the labial surface of a tooth without caries or restorations after which the tooth in question was similarly tested. Momentary pain was produced in the control tooth while the reaction in the tooth with diseased or injured pulp was either that of prolonged pain or negative. Cold was applied to the control and questionable tooth by means of ice or ethyl chloride sprayed on a small piece of gauze. Momentary pain was experienced in the control teeth while in the diseased teeth intense or prolonged pain, relief or no reaction was noted. A negative reaction to cold and heat indicated death of the pulp. Positive reaction to cold and heat was found with inflammatory changes in the pulp. Pain upon application of heat with relief upon cold application seemed to indicate suppurative processes. Since we were of the opinion that thermal tests recorded volumetric changes and the condition of the circulatory elements only, while the electrical tests recorded nerve irritability in the pulp, both methods were used to determine vitality of the tooth in question.

TABLE I

Teeth selected for root therapy

MAXILLARY								MANDIBULAR							
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
0	1	18	57	52	24	49	69	1	2	2	14	17	30	3	1

After a tentative diagnosis of the condition of the pulp had been made using the x-ray and vitality tests, the tooth was isolated with rubber dam. The entire field was then painted with 7% tincture of iodine followed by 70% alcohol. Access to the pulp chamber was established with sterile burs and spoon excavators. The pulp tissue was removed with sterile barbed broaches. A sterile absorbent paper point was placed in the canal and allowed to absorb moisture for 1 minute, then dropped into a tube of hormone broth. Another point was wiped across the field of operation and rubber dam and a third point was removed from the root therapy tray and placed in a tube of hormone broth. After 48 hours incubation at 98°F., the media were examined microscopically. If no organisms were revealed, sterility was assumed. If organisms were found, a loopful of inoculated media was smeared on a blood agar plate to check the hemolytic properties of the organisms, and other portions inoculated into various sugar solutions to check upon reducing properties. By microscopic and cultural studies the identity of the organisms was established. Between visits medicated dressings of beechwood creosote or formocresol were left sealed in the canal using the modified zinc oxide and eugenol cement recommended by Grossman. After 2 sterile cultures were obtained the canal was permanently filled with gutta percha cones or silver points.

FINDINGS AND DISCUSSIONS

Attempts were made to analyze and correlate the vitality test findings with roentgenographic findings and also sterility or infection. A resume of the diagnostic findings can be found in Table II. In the 340 cases, 263 pulps (77.3%) were positive to vitality tests and 77 (22.7%) were negative. Of the vitality

TABLE II

Diagnostic resume

Vitality	Positive (263)	X-ray	Positive (57)
			Negative (206)
		Bacteriologic	Positive (150)
			Negative (113)
	Negative (77)	X-ray	Positive (40)
			Negative (37)
		Bacteriologic	Positive (61)
			Negative (16)
X-ray	Positive (97)	Vitality	Positive (57)
			Negative (40)
		Bacteriologic	Positive (62)
			Negative (35)
	Negative (243)	Vitality	Positive (206)
			Negative (37)
		Bacteriologic	Positive (149)
			Negative (94)
Bacteriologic	Positive (211)	Vitality	Positive (150)
			Negative (61)
		X-ray	Positive (62)
			Negative (149)
	Negative (129)	Vitality	Positive (113)
			Negative (16)
		X-ray	Positive (35)
			Negative (94)

positive pulps, 113 (42.3%) were sterile at first examination while 150 (57.7%) were infected. Although one would expect these teeth to be sterile in most instances, here there were more infected pulps. Of the 77 vitality negative pulps, 61 (76.2%) were infected while 16 (23.8%) were sterile on first check. These findings are more according to expectations.

In correlating the vitality tests with the roentgenographic diagnosis, 206

(79.4%) of the vitality test positive teeth were x-ray negative, while 57 (20.5%) were x-ray positive. The cysts are included in the roentgenographic positive group. This indicates that there is little positive connection between x-ray diagnosis and vitality tests of the teeth, for in 20.5% of our vitality test positive teeth bone changes were demonstrable at the apex. In the 77 teeth testing vitality negative, 40 (52.7%) were x-ray positive, while 37 (47.3%) were x-ray negative. These findings fail to indicate an intimate connection between vitality tests of the pulp and bone changes at the apex.

A total of 243 cases were x-ray negative while 87 were x-ray positive. On first examination of the pulps, 211 (62.2%) were infected while 129 (37.8%) were sterile. Of the group with infected pulps, 149 (71.5%) were x-ray negative and 62 (28.5%) were x-ray positive. Of the 129 sterile pulps, 94 (72.8%) were x-ray

TABLE III

Cysts

CASE NO.	AGE	TOOTH	BACTERIAL FINDINGS	HISTORY OF TRAUMA	BACTERIA PRESENT
1	20	$\lfloor\underline{1}$	Positive	Yes	*Streptococcus viridans*, Kleb-Friedland Bacillus
2	20	$\underline{1}\rfloor$	Positive	Yes	*Streptococcus viridans*
3	30	$\lfloor\underline{2}$	Negative	Yes	—
4	32	$\lfloor\underline{1}$	Positive	Yes	*Staphylococcus aureus*
5	32	$\underline{1}\rfloor$	Positive	Yes	*Staphylococcus aureus*
6	19	$\lfloor\underline{1}$	Negative	Yes	—
7	24	$\lfloor\underline{1}$	Positive	Yes	*Streptococcus viridans*
8	13	$\lfloor\underline{2}$	Negative	Yes	—
9	13	$\lfloor\underline{3}$	Negative	—	—
10	22	$\lfloor\underline{1}$	Negative	Yes	—
11	14	$\underline{1}\rfloor$	Negative	Yes	—
12	14	$\underline{1}\rfloor$	Negative	Yes	—
13	19	$\underline{3}\rfloor$	Positive	Yes	*Staphylococcus albus*
14	24	$\underline{3}\rfloor$	Positive	Yes	*Streptococcus viridans*

negative while 35 (27.2%) were x-ray positive. As with the findings on vitality tests of the pulp, we could show no consistent correlation between infection in the pulp and evidence of bone change in the periapical area. This indicates that bone changes can take place whether the contents of the root canal are sterile or infected. This view is supported by the beliefs of Leriche and Policard (8) who stated that bone resorption may occur by osteolysis. It also reemphasizes the opinion that teeth should not be condemned to extraction in every instance because of the roentgenographic evidence of bone change. Since many such teeth are not a source of danger to the patient, extractions would not benefit the patient in many instances. Grieg (9), Box (10), and others have demonstrated that these bone resorptions and changes can be produced independent of the presence of infection.

The detailed findings for the cystic group are seen in Table III. Among the x-ray positive teeth, 14 were diagnosed as cystic, roentgenographically. This

diagnosis was later verified by histological examination. Of these, 2 had tested vital and 12 non-vital. Both vitality test-positive pulps were also sterile. Five that were negative to vitality tests were sterile while 7 were infected. This group was numerically small and therefore we cannot draw any conclusions from these findings. It is interesting, however, that 2 pulps which tested vital had cystic areas at the apex, diagnosed by x-ray and microscopical study of removed tissue. In both instances the cyst involved 2 adjacent teeth. Both cases followed traumatic injury to the teeth. It is assumed, therefore, that the cysts began to grow from the rest cells of Malassez in the periodontal membranes of the pulpless teeth and, that although the cysts encroached upon the apices of the vital teeth, it pushed the circulatory and nervous elements of the pulps of the vital teeth aside without devitalizing the pulps.

In addition to the attempts to correlate the findings in the x-ray, vitality and bacteriological tests with each other, an endeavor was made to relate the method of pulp exposure with the vitality tests and sterility of the pulp. It was found that of the 286 pulps exposed by caries, 247 (86.3%) tested vitality positive while only 39 (13.7%) were negative. Again in this group, 107 (34.6%) were sterile while 179 (65.4%) were infected upon first bacterial study. This means that in most cases where the dentin is carious down to the pulp chamber, bacteria have preceded the carious process in the dentin, and although many pulps respond to vitality tests under caries, many are at the same time infected.

This is probably an important factor in determining the response of the pulp to conservative pulp treatment of pulp capping and pulpotomy. In the group of pulps exposed by trauma 30 (75%) of the cases were infected while 10 (25%) were sterile. Of these 26 (65%) were negative to vitality tests while 14 (35%) were positive. This group is numerically small and therefore the figures stated do not carry much significance. One would expect the majority of the pulps exposed by sudden, violent traumatic injury to be infected and devitalized for when this trauma is severe enough to fracture the crown of the tooth it may also be intense enough to sever the blood vessels of the pulp at the apex. Likewise since the crown is fractured, the dentin and pulp are probably exposed to the saliva and bacterial flora of the mouth. The detailed bacteriological findings correlated with the x-ray diagnosis and vitality tests can be seen in Table IV.

A variety of organisms were found in the pulps of teeth studied. Forty-one were infected by a non-hemolytic streptococcus. *Streptococcus hemolyticus* was demonstrated in only 1 pulp, that of a 19 year old girl who had recently recovered from scarlet fever. Her only complaint subsequent to her recovery had been anemia. This organism, which is associated with scarletina, may have remained in small numbers in other localities characterized by peculiar circulatory arrangement such as exists in the dental pulp. This is probably an example of the anachoretic effect in pulpitis described by Robinson and Boling (11). In 53 cases *Streptococcus veridans* was present while in 56 pulps *Staphylococcus albus* was found. In 5 pulps *Staphylococcus aureus* was demonstrated. *Lactobacillus acidophilus* was found in only 18 pulps and combinations of bacteria were present in 37 pulps. It is interesting that *L. acidophilus*, which is thought by the Michi-

gan group and others to be one of the etiological agents in caries, was found in only 18 pulps even though most exposures were made by caries. This does not disprove the possible importance of the organism in caries, etiologically, since it may be argued that in deep cavities we have secondary invaders, notably staphylococci and streptococci, predominating. The latter organisms are normal inhabitants of the oral flora and once the opening into the dentin is created by caries, trauma or operation these bacteria can easily invade deeper dental tissues. The vital pulps were primarily infected by non-hemolytic streptococci, *Streptococcus veridans* and *Staphylococcus albus*. The non-vital pulps contained a predominance of staphylococci and combinations of bacteria.

When observed in terms of roentgenographic diagnosis, it was found that in the x-ray positive group *Staphylococcus albus* and combinations of bacteria were most often present while in the negative group *Streptococcus viridans*, *Staphylococcus albus* and non-hemolytic streptococci appeared most often. Over all, those pulps which were vitality test positive and x-ray negative were most commonly

TABLE IV

Bacteria present

	X-RAY DIAGNOSIS		VITALITY RESPONSE	
	Negative	Positive	Negative	Positive
Number of cases	243	97	37	263
Bacteriologic negative	94	35	16	113
Bacteriologic positive	149	62	61	150
Non-hemolytic streptococcus	31	10	9	32
Hemolytic streptococcus	1	0	0	1
Streptococcus viridans	42	11	11	42
Staphylococcus albus	38	18	16	40
Staphylococcus aureus	2	3	3	2
Bacillus acidophilus	13	5	7	11
Bacterial combinations	22	15	15	22

infected by *Streptococcus veridans*, non-hemolytic streptococci and *Staphylococcus albus;* while those which were vitality test negative and x-ray positive were infected by *Staphylococcus albus* and combinations of bacteria.

It should be pointed out that too much emphasis on the importance of the presence of bacteria in the pulp is misleading. Fish and McLean (12) have demonstrated that even though the bacteria can be found in the pulps of the teeth, the neighboring alveolar bone is often sterile. Tunnicliff and Hammond (13) have shown that the presence of bacteria in pulps is not, per se, evidence of infection.

SUMMARY

Three hundred and forty teeth selected for root therapy were subjected to diagnostic procedures. Vitality tests, x-rays and bacteriological tests were made in all cases. Attempts were made to correlate the findings with each other and with those of other investigators.

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STUDIES ON THE INCIDENCE AND CAUSE OF DENTAL DEFECTS IN CHILDREN[1]

III. Gingivitis

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The condition known as gingivitis, signifying an inflammation of the gingivae of various degrees of severity, is a universally distributed dental affliction. While entire groups of inhabitants of some parts of the world have been found free from caries, no similar reports have been submitted regarding gingivitis. Conditions in the past were apparently not much different, and gingival troubles are reported to exist even today in primitive people. Studies on the incidence of gingivitis in children should command an interest equal to that pertaining to caries. Therefore it is well that the relation of this disorder to dental decay should be considered and the various views as to its cause be discussed.

McCall (1) asserted that gingivitis is almost universal in the children attending the Guggenheim Clinic in New York. In his opinion the ailment is as common as caries. He considered that the etiological factors in these juvenile cases are dental decay and occlusal trauma. Warwick (2) associated simple gingivitis with mouth breathing. When the mouth is open the negative pressure (normally present) is absent and the gingivae become hypertrophied and edematous. Sömjen (3) ascribed bleeding gingivae to the same cause and cited a case which points strongly to such relationship. Oddlaug (4) linked gingivitis to an insufficiency of vitamin C in the diet. His conclusions are based on an investigation of school children of Arnes and Finnmark and a group of Lappish children. The latter, with a greater consumption of foods rich in vitamin C, showed a lower incidence of both caries and gingivitis. It is necessary briefly to report that my field and clinical observations failed to substantiate the contention that the prevention or arrest of gingivitis and caries is attained through adequate vitamin C intake.

Klatsky (5) noted, through studies carried out on hundreds of patients, that individuals who eat harder foods invite normal function of the masticatory apparatus. On the other hand, those living on soft foods provoke less strenuous masticatory effort and, as a result show more prominent accumulations of tartar around the gingival line, a condition largely responsible for the occurrence of periodontoclasia. Klatsky's findings are corroborated by Carque (6), Wallace (7), and others. In this connection it may be remarked that Belding and Belding (8) place special emphasis on the fact that periodontoclasia in young children is practically unknown although the teeth and gingivae are but little exercised.

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My observations confirm this conclusion. Recently Gerson (9) related diseases of the gingivae and teeth to sticky carbohydrate masses adhering to the surfaces of the teeth. He considered white flour the commonest offender and recommended its replacement by fibrous self-cleansing foods—whole wheat bread being one of the most important. Gerson's position appears to a certain degree plausible. However, convincing experimental evidence (pertaining to a large number of subjects) is needed in order to concede his point of view. In the 102 cases which the author watched, the self-cleansing qualities of whole-wheat bread were not apparent and testimony of its beneficial influence on the prevention or arrest of gingivitis and caries was absent. Yet I believe that the fibrous food theory deserves special attention. To form a definite judgment upon the data accumulated so far would be rash and further investigations on this subject are in progress.

The part played by the endocrines in the chemistry of the human body and in the causation of dental abnormalities assumes an ever increasing importance. According to Reynolds (10) the structure of the gingivae, as well as that of the alveolar process, may be determined by these glands. Brucker (11, 12) has observed poor gingival conditions associated with disturbances of the thyroid, the pituitary body, the gonads, and the islands of Langerhans in the pancreas. Other investigators report similar findings.

At this point let us consider various accounts of the incidence of gingivitis. My studies show that a connection between caries and this disease is difficult to establish. For example, Sterling (13) (Atlanta, Georgia), and Clements and Kirkpatrick (14) (N. S. Wales, Australia), have presented data showing a considerable predominance of caries over gingivitis. On the other hand, Barnes (15) (Tristan da Cunha), Kirkpatrick (16) (the island of Manus), Staz (17) (Bantu of S. Africa), and Campbell (18, 19) (aborigines of Australia) point to a preponderance of gingivitis over caries.

The merit of the toothbrush as an aid in preventing dental ills, including gingivitis, appears to be a debatable issue. However, information suggesting its usefulness is certainly in existence. For example, Saunders and Taylor (20) report the presence of gingivitis in 34 of the 36 Maori children they studied. These people do not use the toothbrush.

DATA

A study was made of 1,634 public school pupils of Newark, New Jersey, whose ages ranged from 4 to 16 years. Their socio-economic levels varied; 14 nationalities were included; there were 7 negro girls in the group (Table I).

It was deemed important to divide those investigated into 3 groups: first, those with "clean" teeth; second, those whose teeth were "fair"; and, third those who had "dirty" teeth. The term "clean" teeth denotes a freedom of these organs from stains and food; "fair" teeth signifies the presence of stains and especially food on some of the teeth; "dirty" teeth indicates stains and excessive debris on all teeth.

The classification of those afflicted with gingivitis was based primarily on a careful visual examination. The mirror, explorer, college pliers, scalers, and

cotton rolls were used in the procedures. Pupils revealing a mild marginal inflammation with a limited tendency to bleeding and with a history of occasional hemorrhage on brushing were declared "simple" or "slight" cases of gingivitis. Those disclosing a deeper seated inflammation with "puffiness" of the gingival margins and with interstitial involvement were classed as cases of "severe" gingivitis. In them, a change in the outline of the gingivae and a tendency to bleed at the slightest provocation were noted. In order to ascertain a possible exist-

TABLE I

Condition of gingivae in 819 boys and 815 girls studied. Seven negro girls included

	HEALTHY GINGIVAE	SLIGHT GINGIVITIS	SEVERE GINGIVITIS
Number of boys	735	72	12
Number of girls	757	47	11
Total	1492	119	23

TABLE II

"Clean" teeth and the incidence of gingivitis

	WHITE		NEGRO GIRLS	TOTAL
	Boys	Girls		
No. with clean teeth and healthy gingivae	345	402	4	751
No. with clean teeth and slight gingivitis	8	14	0	22
No. with clean teeth and severe gingivitis	0	0	0	0

TABLE III

"Fair" teeth and the incidence of gingivitis

	WHITE		NEGRO GIRLS	TOTAL
	Boys	Girls		
No. with fair teeth and healthy gingivae	387	348	3	738
No. with fair teeth and slight gingivitis	59	32	0	91
No. with fair teeth and severe gingivitis	8	10	0	18

ence of Vincent's infection in this group, their examination was augmented by the taking of smears from the depths of the gingival crevices.

Of the total number of pupils studied, 773 had clean teeth (Table II). In them, 22 cases of slight gingivitis were registered, 14 in girls and 8 in boys. Eight hundred and forty-seven had teeth in a "fair" state of cleanliness, 59 boys and 32 girls in this group displayed slight gingivitis, and 18 others, 8 boys and 10 girls, disclosed inflammation of the gums of a vastly more serious nature (Table III). An

important point is that the 4 negro girls revealing clean teeth, and also the 3 with fair teeth were the possessors of healthy gums. Of the 14 children with dirty teeth, 6 had slight gingivitis and 5 others inflammation of a severe character (Table IV).

The fact stands out that the data amassed here substantiate the views of those who uphold the doctrine that the state of teeth is correlated with that of the gingivae. That is, good gingivae are most apt to be found associated with clean teeth, fair gingivae with teeth that are fairly clean, and diseased gingivae with dirty teeth. No one denies that gingival diseases may develop despite meticulous observance of good oral hygiene. An explanation for their etiology must be

TABLE IV

"Dirty" teeth and the incidence of gingivitis

	WHITE		NEGRO GIRLS	TOTAL
	Boys	Girls		
No. with dirty teeth and healthy gingivae	3	0	0	3
No. with dirty teeth and slight gingivitis	5	1	0	6
No. with dirty teeth and severe gingivitis	4	1	0	5

TABLE V

Caries and gingivitis. Incidence of caries in pupils possessing healthy gingivae and in those having slight and severe gingivitis

	NUMBER EXAMINED		NUMBER OF CAVITIES PRESENT		AVERAGE NO. CAVITIES PER CHILD	
	Boys	Girls	Boys	Girls	Boys	Girls
Healthy gingivae	735	757	1846	2590	2.5	3.3
Slight gingivitis	72	47	239	176	3.3	3.7
Severe gingivitis	12	11	48	55	4.0	5.0
Total	819	815	2133	2821		

sought in systemic, not in local predisposing causes. We have already alluded to the part played by the glands of internal secretion in the etiology of gingivitis. Other systemic disturbances of non-endocrine origin may also act as predisposing causes.

I have noticed instances in young children where, in spite of a lack of oral hygiene, the condition of the gingivae appeared satisfactory. Clinical and follow-up observations on many such pupils have convinced me that their state of health is temporary. As time goes on, the effects of lack of oral care are definitely demonstrated and the true consequences of neglect become manifest in later years.

Of the 1,634 pupils studied, 142 (58 girls and 84 boys) had gingivitis, in other words, 2 out of every 23 students suffered from this condition. Of these, 72 boys and 47 girls showed slight gingivitis. An average of a little over 3 cavities per

child was noted in those of both sexes in this stage of the disorder. Twenty-three of those investigated, 12 boys and 11 girls, had severe gingivitis. In them a higher incidence of dental decay was revealed: 4 cavities each were registered for the boys and 5 cavities each for the girls (Table V).

DISCUSSION

There was a higher incidence of carious teeth in the children with gingivitis than in those who were free from the disease, yet it is difficult to believe that caries was the factor responsible for the initiation of the disorder. The final answer to this problem lies in further investigation on the subject.

A fact which calls for particular attention is that the study of these Newark pupils revealed a considerably lower incidence of gingivitis (8.7 per cent) than that reported in the aborigine children of Cockatoo Creek, Central Australia (27 per cent); lower than in the group of New South Wales children reported by Clements and Kirkpatrick (61 per cent), the Maori children reported by Saunders and Taylor (20) (over 94 per cent), and the children of Colombo, Ceylon, as reported by Balendra (21, 22) who stated that gingivitis and pyorrhea occurred in about 20 per cent of those he examined. A strikingly lower incidence was exhibited in comparison with McCall's findings of 98 per cent of gingivitis.

The possibility suggests itself that colored children are less susceptible to gingival disease than white children. For example, Sterling reported that 3 per cent of the 5,000 colored children she examined revealed gingivitis. My own daily clinical and field examinations show that gingivitis is less prevalent in colored pupils. Further studies are necessary for the understanding of the inherent qualities which make this condition possible.

Warm climates seem to exert no inhibitory influence on the incidence of the disease, for a higher proportion of children inhabiting warm territories have been reported with gingivitis than in the Newark children.

That correct habits of oral hygiene may have played a contributory part in the promotion of better gingival condition in the Newark pupils appears distinctly probable. There are positive grounds for this conclusion.

CONCLUSIONS

The total findings indicate that no correlation between the incidence of caries and that of gingivitis was demonstrated. The number of pupils with inflammation of the gingivae was much smaller than that of those with dental caries (active caries in 70 per cent; gingivitis in 8.7 per cent). In connection with this it is well to recall that Kirkpatrick, Barnes, Staz, Campbell and others found the incidence of gingivitis greater than that of caries. This indicates that the theory of relationship between the 2 disorders is unfounded and, to my knowledge, such a relationship has not been established.

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STUDIES ON THE INCIDENCE AND CAUSE OF DENTAL DEFECTS IN CHILDREN[1]

IV. Malocclusion

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The relation of malocclusion to the incidence of dental caries has received the attention of numerous investigators. That faulty arrangement or relation of the teeth plays an important part in the production of dental decay is the opinion of many students of the problem, who maintain that normal occlusion is the important factor in immunity to caries. Such for instance is the view of Weston (1), deputy school medical officer of Shropshire. In reporting on a group of children "who were naturally free from caries," the point is emphasized that over 97.8 per cent disclosed good dental occlusion. Radcliffe (2), school dentist of Keighley, England, presented an interesting summary on 30 children she has studied. Fifteen of those under her observation had perfectly sound teeth; the other 15 showed caries in nearly every tooth. Most of the children in whom caries was most severe had narrowed arches and enlarged tonsils. In those immune to the disease occlusion was normal. The findings of Ferguson (3) substantiate Weston's and Radcliffe's contentions. A low total of 26 cases of malocclusion was noted among the 2,257 pupils he examined in the public schools of American Samoa (18 in the male and 8 in the female). Ninety per cent of those studied were free from caries. Yet Sampson (4), in Tristan da Cunha, found that most of these islanders had perfect teeth. Nevertheless, edge to edge bite was present in a large proportion of them and one case of open bite was also reported. McRae (5), who examined a mixed group of 3,188 white and negro children, found that although the white children had better occlusion than the negroes, the latter showed a lower incidence of caries. In his studies Staz (6) noted that those of his subjects with "medium arches" seemed to have fewer cavities than those with "well-formed" arches. Thus it would appear that the shape of the dental arches, although probably involved in the promotion of caries, is not the only factor conducive to dental disorders.

Gilmour (7) inferred that impaired health of the mother during pregnancy may be responsible for the narrowed arches so often encountered in children. In his opinion, a tendency to malocclusion may be congenital or inherited. A similar view was held by Mashankar (8) whose observations are based on a study of 4,365 children of Akola, India. Lubowitz (9) and Nord (10) related experiences to show that open bite may be hereditary. Munblatt's (11) studies add weight to the contention that heredity plays an important rôle in facial growth as well as in the production of malocclusion.

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Steadman (12) maintained that modern civilization with its trend toward the consumption of sugary, tooth-adhering carbohydrate food articles is mainly responsible for caries, periodontosis and malocclusion. His studies of a group of skulls of Tasmanian aborigines revealed in them an almost complete absence of dental caries and periodontosis and a higher average of normal occlusion than in civilized man. He pointed out that the factor responsible for their well-formed jaws was the consumption of such foods as lobsters, oysters, fruits, berries, birds and their eggs, and such animals as they were able to kill.

It has been intimated by other investigators [Price (13), Badanes and Barodneck (14), and others] that refined foods and modern dietary habits in general are to blame for our present day dental ailments. Thus civilization attains a new and vicious distinction. However, we are bound to remember that manifestations of malocclusion existed long before contact with civilization was possible. They have been in existence as long as man has been man.

Bradley (15) expressed the opinion that occlusal stress is one of the causes of malocclusion and all forms of caries except that of pits and fissures. Proximal caries in the incisal region is "associated with stress from incisal contact when the jaws are in centric relation or from incisal interference during mastication." He further stated that caries in the proximal surfaces of the posterior teeth is due to stress reception by one or both teeth resulting from "interference with the occlusal curve." Bradley's theory is important and deserves special consideration. His additional conclusions are commended to the attention of students of the problem. The influence of occlusal trauma on the causation of caries has been reported by a number of other investigators. It is important to emphasize the view of Abrahams (16), who was convinced that responsible for this occlusal trauma is the present day milk craze and the constant feeding of milk to growing children. This so-called perfect food fails to stimulate masticatory function and normal development of the dental arches. The indications are that disturbed occlusion is promoted instead. Wallace (17) also considered milk an important factor in the etiology of dental irregularities and their sequellae. When the growing child attains the age of $2\frac{1}{2}$ years the teeth, the jaws, in fact the entire digestive apparatus are in need of food other than milk which was suitable in infancy. Day to day my own field and clinical investigations emphasize this situation.

DATA

A group of 1668 white school children (863 boys and 805 girls) of Newark, New Jersey were studied. Their ages ranged from 5 to 15 years and their socio-economic status was above the average.

The pupils investigated were divided into 3 groups: first, those with malocclusion of either the permanent or the deciduous teeth (no mixed dentition was present in any of them); second, those with malocclusion of the transitional or mixed dentition; and third, those who were free from malocclusion.

The children comprising the first group will be referred to as having "true" malocclusion. The pupils in the second group will be alluded to as having "malocclusion of the transitional dentition." While a substantial number of

nationalities were included among those investigated, they were separated into 2 sub-groups; namely, Jewish and non-Jewish.

Of the total number of pupils examined, 182 boys and 195 girls showed true malocclusion and 324 children had malocclusion of the transitional dentition. The balance, 967 children, were free from any form of these anomalies (Table I). The classification of children affected with malocclusion of the permanent or of the transitional dentition (in the latter cases where the 4 first permanent molars were fully erupted) was based on standards set forth by Angle. The three classes (Class I, Class II, Class III) were determined by the mesio-distal relationship of the upper and lower arches as signified by the interlocking of the upper and lower first permanent molars when the jaws were brought into contact. Briefly reiterated: Class I is characterized by a normal mesio-distal relationship of the upper and lower arches, with malocclusion of the anterior teeth; in Class II the lower arch and teeth are distal to normal in their relation to the upper; in Class III the lower jaw and teeth are mesial to normal in their relation to the upper. The classification of children affected with malocclusion of the deciduous dentition (with no first permanent molars erupted) was based on the relationship of the upper and lower teeth when the jaws were brought together.

TABLE I

Incidence of malocclusion in a group of 1668 white boys and girls

	WHITE BOYS	WHITE GIRLS	TOTAL
Number of pupils examined	863	805	1668
Number of pupils with malocclusion of the transitional dentition	174	150	324
Number of pupils with true malocclusion	182	195	377

The locking of the cusps and the harmonious relation of the jaws were the factors considered.

The diagnosis of malocclusion was based almost solely upon a most careful and exacting clinical examination. In some instances this was supplemented by the taking of impressions, the making of plaster reproductions, and radiographic adjuncts. By constantly considering the significance of the three classes and their divisions and subdivisions, the possibility of error in diagnosis and classification was distinctly remote.

It is important to note that 57 children (28 boys and 29 girls) with true malocclusion (of either the permanent or the deciduous teeth) were free from caries, that is, they had no past history or present indication of decay. Forty-nine pupils (29 boys and 20 girls) with malocclusion of the transitional dentition were also free from caries. Of the latter number, 38 showed deep overbite, eight open bite, and three edge-to-edge bite (Tables II and III).

The relation of nationality to malocclusion and caries was limited to a study of only 1,268 of the total number examined. As the nationality of the others could not be definitely ascertained it was considered best not to include them. The incidence of true malocclusion and also that of the transitional dentition was

slightly greater in the non-Jewish sub-groups. The element responsible for the increased susceptibility in the non-Jewish could not be determined. If the subject of nationality and incidence of malocclusion of the transitional dentition is further considered, it will be seen that more Jewish than non-Jewish children exhibited overbite. However, more non-Jewish boys and girls showed open bite. The proportion of children with edge-to-edge bite was slightly higher in the non-Jewish subjects. As to the occurrence of true malocclusion in the Jewish and non-Jewish boys, the following facts were revealed. Class II cases

TABLE II

Malocclusion and freedom from caries

	MALOCCLUSION OF THE TRANSITIONAL DENTITION			TRUE MALOCCLUSION		
				Class		
	Over bite	Open bite	Edge to edge bite	I	II	III
Number of boys	22	6	1	11	17	0
Number of girls	16	2	2	*		

* 29 girls had true malocclusion and were free from caries. However, no classification was attempted here.

TABLE III

Malocclusion and caries

	WITH TRUE MALOCCLUSION		WITH MALOCCLUSION OF THE TRANSITIONAL DENTITION		WITH NO MALOCCLUSION	
	Boys	Girls	Boys	Girls	Boys	Girls
Number of pupils	182	195	174	150	507	460
Number of cavities	772	842	715	631	1218	1341
Average number cavities per child	4.3	4.3	4.1	4.2	2.4	2.9

predominated in both groups, the next in order as to distribution being the Class I cases. The proportion of Class III cases was about the same in both aggregations of boys studied.

In this study of true malocclusion a complete classification was confined to the boys, the various class manifestations noted in the girls being grouped together. Cases of edge-to-edge bite were placed in a group by themselves. This type of anomaly was not observed in any of the Jewish boys inspected. More non-Jewish than Jewish girls with true malocclusion had edge-to-edge bite.

No attempt was made to segregate strictly according to class those showing malocclusion of the transitional dentition. Wolfson (18) expressed the belief that "very many of the so-called deviations from normal occlusion which we observe in young children may be merely stages in development." Don Chalmers Lyon (19) suggested that many cases of deep over-bite "correct themselves" in later years (between 12 and 14). He advised against treatment until

the premolar teeth have assumed their places in the arches. Taking all these deliberations into account, it should be emphasized that even though malocclusion of the transitional dentition may correct itself later in life its influence, while present, on the occurrence of caries is evident.

The incidence of caries in the non-Jewish boys and girls showing malocclusion of the transitional dentition was somewhat higher than in the Jewish boys and girls (Table VII). It was difficult to decide whether the element of nationality was accountable for this circumstance. The incidence of dental decay in those with true malocclusion was higher in the non-Jewish boys showing Class I, Class II and Class III malocclusion than in the Jewish boys in the corresponding

TABLE IV

Incidence of malocclusion at various ages

Ages	5	6	7	8	9	10	11	12	13	14	15
True malocclusion	6	3					5	81	89	96	97
Malocclusion of the transitional dentition			33	73	77	82	49	10			

TABLE V

Incidence of pupils with malposed individual permanent teeth

Number of pupils with malpositions in the upper arch	95
Number of pupils with malpositions in the lower arch	78
Number of pupils with malpositions in both arches	19

TABLE VI

Incidence of malposed individual permanent teeth

	Upper right							Upper left						
Tooth*	7	6	5	4	3	2	1	1	2	3	4	5	6	7
No. malposed	0	0	5	1	8	42	21	27	27	8	3	7	1	0
Tooth	7	6	5	4	3	2	1	1	2	3	4	5	6	7
No. malposed	0	0	3	0	6	45	32	37	34	4	0	1	0	0

* Palmer's notation.

groups. It may be added that the incidence of caries in the girls afflicted with true malocclusion was higher in the non-Jewish than in the Jewish. Again, the element involved in the production of this state was difficult to decide. Further studies along this line are indicated in order to establish the influence, if any, of nationality on the occurrence of caries as related to disturbances of occlusion. Anthropological factors and their possible relationship to an increased resistance to dental decay under these circumstances in the Jewish sub-group deserve consideration.

It is noted in Table IV that the age at which malocclusion of the transitional dentition was most frequently manifested was 10 years (82 cases). The age at which true malocclusion was found to predominate was 15 years (97 cases).

In a discussion of malocclusion the subject of individual tooth malpositions must receive its share of attention. While such single malpositions may not distort the general relationship of the upper and lower dental arches, they often give rise to complications which may become severe. Gingivitis of various degrees, loosening of the involved teeth, injury to the opposing tooth are but a few of the consequences that may be noted. A total of 192 pupils (100 boys

TABLE VII

Malocclusion of transitional dentition. Incidence of caries in various types manifested in Jewish and non-Jewish boys and girls

	JEWISH			NON-JEWISH		
	Over bite	Open bite	Edge to edge bite	Over bite	Open bite	Edge to edge bite
Number of pupils	148	33	4	78	54	6
Number of cavities present	492	135	22	363	312	21
Average number of cavities per child	3.4	4.1	5.5	4.6	5.9	3.5

TABLE VIII

True malocclusion. Incidence of caries in various class manifestations in Jewish and non-Jewish boys

	JEWISH				NON-JEWISH			
	Edge to edge bite	Class			Edge to edge bite	Class		
		I	II	III		I	II	III
Number of pupils		37	54	5	6	31	45	4
Number of cavities present		135	190	28	23	133	226	33
Average number of cavities per child		3.6	3.5	5.3	3.8	4.3	5.0	8.2

TABLE IX

True malocclusion. Incidence of caries in various class manifestations in Jewish and non-Jewish girls

	JEWISH		NON-JEWISH	
	Edge to edge bite	True (various classes)	Edge to edge bite	True (various classes)
Number of pupils	6	96	10	80
Number of cavities present	35	352	45	392
Average number of cavities per child	5.8	3.6	5.5	4.7

and 92 girls) showed a total of 312 malposed permanent teeth. There were more such malpositions in the lower than in the upper arch (Table V). Malpositions in both arches were rare. The permanent tooth most frequently affected was the lower right second incisor, followed by the upper right second incisor and the lower left first incisor in consecutive order (Table VI). Four girls with complete complements of deciduous teeth showed malposition of the lower right

second incisor; in 3 others marked malalignment of the upper left second incisor was noted.

CONCLUSIONS

The findings accumulated indicate that the incidence of caries in children having malocclusion of the transitional dentition was substantially higher than in those not disturbed by any form of the disorder (Table III). Also the incidence of dental decay in those having true malocclusion was higher in the boys and girls so afflicted than in the children who were free from malocclusion. Nineteen per cent of the total number of pupils examined had true malocclusion and in them 29 per cent of the total number of cavities was registered. The influence of malocclusion on the incidence of caries has thus been shown.

This study also brought to light the fact that non-Jewish children afflicted with the condition showed a definitely higher incidence of caries than Jewish boys and girls affected with the disorder, whether of the deciduous, permanent or transitional dentition (Tables VII, VIII, IX). Malocclusion of the deciduous teeth was noted in 9 pupils, all boys. Children free from caries were observed in both groups, suggesting the conclusion that where caries resistance exists no influences, of whatever type, are effective in its initiation.

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HEAT IMPACT OF REVOLVING INSTRUMENTS ON VITAL DENTIN TUBULES[1]

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The application of a fine stream of 100°F. water in "drilling" or grinding teeth during cavity preparation stabilizes the temperature of both dentin and revolving instruments and thereby prevents more than half the usual pain from operative treatment (*fig. 1*). It would seem probable that frictional heat is an important factor in operative pain.[2]

There is considerable literature on heat trauma, but the most comprehensive work was by Jeserich (1). He gave 1°F. to 25°F. as the range of thermal increase for various revolving dental instruments and explained that the variation depended upon size, sharpness, speed, amount and type of pressure, and whether the operation is performed in a dry, moist, or wet field. Jeserich also stated "... it is possible to operate in a dry field with steel burs with slight thermal increase if" the precautions of size, sharpness, speed and pressure are exercised. It is with this latter statement especially, that this paper takes exception.

Bronner reported 10°C. (18°F.) rises, but only during definite abuse (2). Bodecker (3) deplored heat from rapidly revolving instruments and connected it with pain. He produced a rise of 27°F. with a $\frac{5}{8}$ in. carborundum stone used with moderate pressure, continuously for 45 seconds, prolonged grinding that could hardly be recommended. Wilson (4) confirmed Bodecker's experiments but found a rise of 5° to 60°F. depending upon the technique of cutting, the higher figures during abuse. All these and many other reports show temperature rises of less than 30°F. during normal operating, well within "the thermal tolerance zone"[3] of living dentin, yet other writings link pain with frictional heat. Thomas (5) reported that the use of chilled burs (no frictional heat) diminished or eliminated pain in 76.8 per cent of his subjects.

Previous heat measurements, such as Jeserich's, Bronner's Wilson's, and Bodecker's were made to determine the temperature changes of the enamel, dentin, and pulp during the grinding of tooth substance in operative treatment. Throughout this work much importance seemed to be given to the general temperature of the tooth and pulp, with pulp trauma and death as possible sequellae.

[1] Presented at a meeting of the New York Section, International Association for Dental Research, N. Y., January 28, 1943 (*J. D. Res.*, **22**: 165, April, 1943). Received for publication February 27, 1943.

[2] Clinical work over a six year period was by our graduate group: Leon Lieber, D.M.D., Morris Gudwin, D.M.D., Jack R. Kopely, D.D.S., Chester J. Henschel, D.D.S.

[3] For this phrase I am grateful to Dr. Edward J. Ryan.

This concept is inadequate for normal operative procedures, and applies only during operative abuse.

In measuring the increase in enamel, dentin, or pulp temperature during opertive procedures, the usual method is to insert a thermocouple into the pulp chamber or tooth substance and grind within a short distance of the measuring instrument. The recordings made in this way were inadequate because dentin and enamel are good thermal insulators (1). This property retards the penetration of frictional heat into the tooth and confines it largely to the thin layer of dentin in contact with the bur. Most of this energy escapes along paths of less resistance than that of the tooth—into adjacent moisture, air, and into the bur or grinding instrument itself, although all the heat which results from the

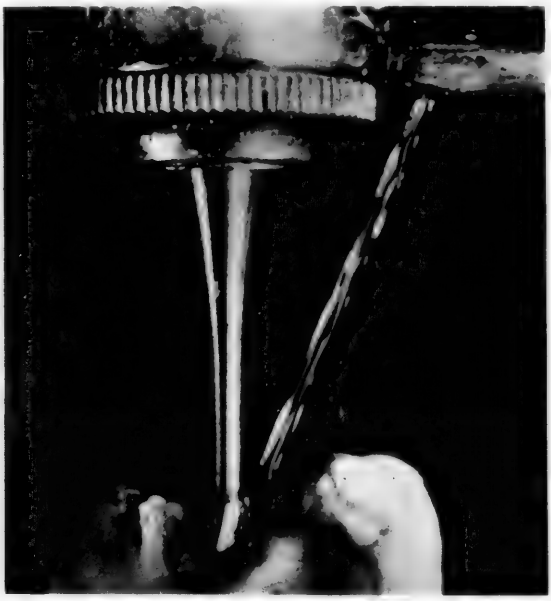

FIG. 1. Potential frictional heat of #34 steel bur is being absorbed by stream of 100°F. water, electrically synchronized with dental engine foot controller.

frictional contact of the bur is focused upon a small surface spot of exposed dentin, it escapes in all directions, in all three planes, least of all in the direction of the conventionally placed thermocouple. The reason why the thermocouple gave only a limited indication of the generation of frictional heat is therefore evident.

Our clinical observations suggested some error in the minimal heat recordings of previous investigators. Similar doubts should be shared by anyone who has felt the appalling frictional heat from a dry bur, as in carving or cutting a groove when a metal inlay is held in the fingers.

This paper is concerned with thermal measurements during skillful treatment, not during operative abuse, with special emphasis on painlessness (6). It has

been recognized that the pulp and living unmetamorphosed dentin (7) are not only extremely sensitive end organs but are especially irritated by thermal changes (8). Jeserich stated (1) that although dentin is a good insulator, thermal changes are felt instantly because of the extreme sensitivity of the dentin fibrils. In my opinion, it is because of this that temperature changes beyond the limits of the "thermal tolerance zone" (9) elicit pain.

Effort is directed at measuring the quantity of thermal energy focused or impacted upon the surface of the cut, open ends of the dentin tubules in actual contact with the bur not with the gross temperature rise of the tooth. My hypothesis is that the concentration of a few calories (as from small burs) on a small area of the odontoblastic processes will usually result in a sensation which the brain interprets as pain. It appears that the surface dentin is burned in much the same way that a small, hot instrument would burn the skin of one's hand without appreciably increasing the temperature of the entire hand. It would seem probable that the actual temperature changes which cause dental pain are more subtle and delicate than had been heretofore realized. The pain arises not at the pulp, but at the small area of bur contact where the temperature rise produces pain much the same as an explorer on vital dentin at its point of contact. Therefore it is of utmost importance to study the temperature rise here, not at the pulp.

It was intended to measure the total caloric production of heat energy by our revolving instruments at "normal" operating speeds and pressure, using this data to calculate the temperature of the actual cut dentin surface and that of the steel blades of the bur. Although nearly all of this heat energy is radiated away and lost almost instantly, during a fraction of its existence all its potency is focused upon between 15,000 and 300,000 dentin tubules, (accepting 30,000 tubules per $mm.^2$) depending upon the size of the bur. The effect of any one portion of this caloric energy is necessarily momentary, but since its generation is continuous it persists as long as the bur revolves. This heat can be measured if water of a known temperature is poured over the frictional surface and its temperature rise noted. Caloric determination then becomes quite simple.

METHOD

Water was used as a heat absorbent at the rate of 8.0 cc per minute, and at a known temperature. It was passed over the working area of the bur or stone while grinding a recently extracted tooth which was placed above the funnel-like opening into an "L" shaped glass tube (*fig. 2*). This tube was insulated by a dead air space, and in the lower portion was placed the thermometer bulb. The water had to pass around the thermometer before draining off. The entire apparatus was mounted upon a spring scale to record operating pressure. The engine foot control was set at second speed, about 2,000 r.p.m. and the temperature of the water cascading from the dentin before, during, and after grinding was recorded to circumvent the influence of room temperature on the water. With the fluid stream, engine speed and pressure need not be limited, and thermal abuse is impossible. However, in an attempt to simulate the conditions

of careful, skillful, operative dentistry, these experiments were conducted under conditions of normal operative restraint.

In the first series of recordings new #558 cross cut cylindrical steel burs were used on recently extracted teeth according to the general plan. This type and size was selected as one of the smaller and gentler varieties which, from the thermal point of view, had been considered "least offensive" (3). However, the final practical interpretation of our experiments indicated that from the "thermal-pain" point of view, there was little difference between the #558 and the smaller inverted cone and round burs, or whether the entire or only part of the bur was used.

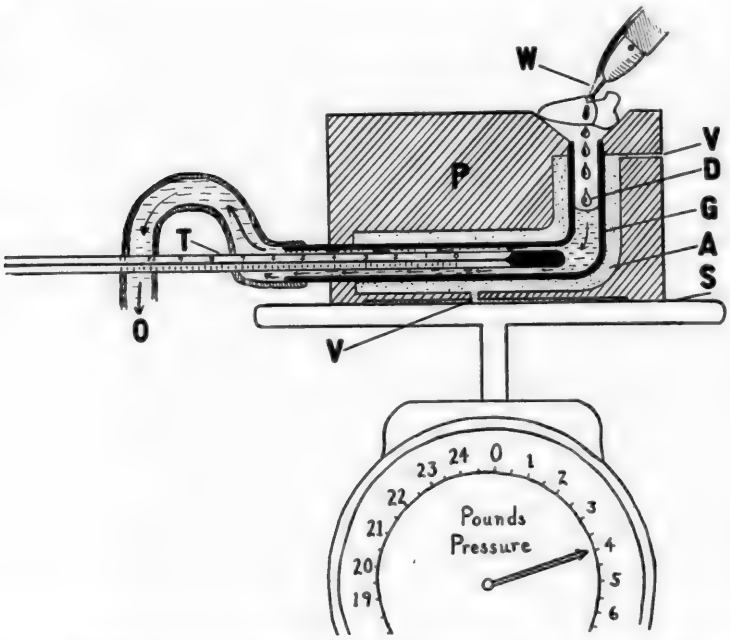

FIG. 2. *Experimental apparatus for caloric determination.* W—water stream (8 cc. per minute), D—drippings of water stream from frictional area, G—glass tube, A—insulating air space, T—mercury thermometer (0–100°C., calibrated in fifths), P—plaster block, O—overflow from waterstream, V—air vents, S—spring scale.

EXPERIMENTAL

Thermal rise: The average rise in temperature during the first experiment was 2.5°C. which showed that the #558 bur at 2,000 r.p.m. and 4 lbs. pressure generated frictional heat amounting to 20 small calories per minute, 8 cc. water heated 2.5°C. The average #558 bur head is a cylinder 3.8 mm. long and 1.2 mm. in diameter, with a 360° cylindrical lateral cutting area of about 14 square mm. Only 180 degrees, or one side of a cylindrical bur can be used for cutting at one time; therefore the working surface area of the #558 bur may be considered to be

7 square mm. From this source we may arbitrarily estimate potent heat penetration into dentin to be about 0.5 mm. or less in depth. Thus the 20 calories may be applied to this semi-circular section of dentin in contact with the bur, and measuring 3.8 x 1.8 (bur length x ½ circumference) x 0.5 (the depth or thickness), which equals about 3.5 cu. mm. The length of time that the bur cuts, rubs, and heats this section of tooth depends upon two factors: 1. the period of each intermittent application of the bur, and 2. the time required to cut and destroy this amount of tooth structure. Considering both, a frictional period of one second would be a conservative estimate. Since 20 calories are generated per minute, in 1 second the heat developed amounts to .33 calories, which if applied to 3.5 cu. mm. of water (the estimated volume of dentin heated) would result in a rise of 94°C. By other experiments the specific gravity of dentin was found to be 2.11, which was similar to the 2.14 of Manly, Hodge, and Ange (10). Therefore the rise of 94°C. must be divided by 2.1, since the higher density of dentin offers increased weight to heat per volume as compared with water. The resultant figure of 45°C. is next divided by the specific heat of dentin, 0.3,[4] which yields 150°C. or 270°F. This is the thermal rise focused upon 3.5 cu. mm. which contain about 105,000 dentin tubules. After the pain is caused, the energy escapes and is lost (*figs. 3* and *4*). The formula for estimating the thermal rise from a revolving instrument on the surface dentin may be expressed thus:

formula:	calories per second	×	$\frac{1{,}000}{0.5 \times \text{area dentin contacted in mm.}}$	÷	Spec. gravity of dentin	÷	Spec. heat of dentin	=	Estimated Centigrade rise
more simply:	n	×	$\frac{1{,}000}{\text{am't. dentin heated}}$	÷	S.G.	÷	S.H.	=	°C.
example:	0.33	×	$\frac{1{,}000}{3.5}$	÷	2.1	÷	0.3	=	150°C. or 270°F.

Cutting area: The experiments were continued under altered conditions, such as may occur during the preparation of a cavity. A new #558 bur was used

[4] Since a search of the available literature failed to disclose any specific heat determination, the author arrived at the figure of 0.3 by a method exemplified by the following experiment. 50 grams of dentin at room temperature was dropped into a vacuum bottle which already held 100 g. of water at 53.4°C. (stabilized). The drop in temperature of the water indicated the calories necessary to heat 50 g. dentin from room temperature of 23.0°C. to the final mutual dentin-water temperature of 49.15°C. The figures were adjusted by ascertaining the slow but regular heat loss when closed and the loss when opening and closing the vacuum bottle for the 15 seconds interval necessary to introduce the dentin. Thus, 14.92 calories were required to heat 50 grams of dentin one Centigrade: 14.92 ÷ 50 = .298. This method of mixtures was checked by ascertaining the specific heats of other substances of known values—glass, iron, gold, water. Fresh cortical bone was also checked, as chemical analysis disclosed its similarity to dentin. This bone also had a specific gravity of 2.1 and a specific heat of 0.3. Since the specific heat of dentin necessarily varies with age, sex, and other influencing factors, a more accurate determination than the above seemed inadvisable. The figure of 0.3 represents an average of 30 separate tests.

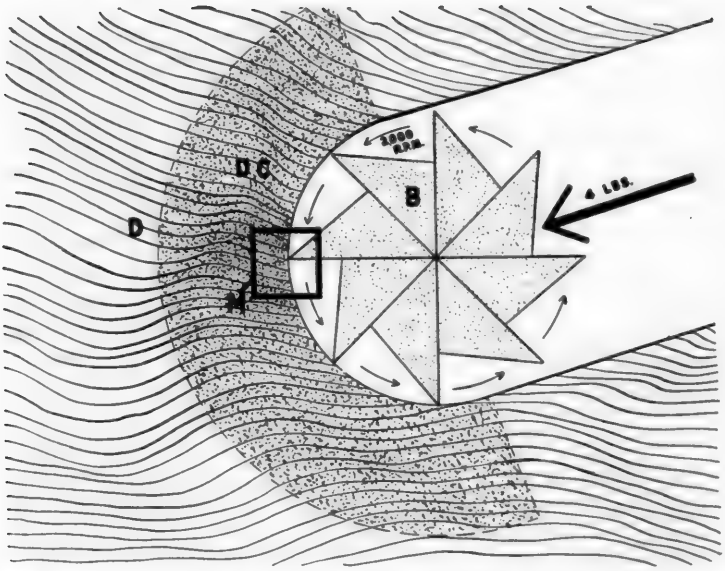

FIG. 3. *Schematic cross-section diagram of #558 steel cross cut fissure bur at 4 pounds operating pressure and 2,000 r.p.m., applied to dentin.* B—#558 steel bur, cross section, bur head 1.2 mm. diameter, 3.8 mm. in length, blades in contact with dentin, DC—0.5 mm. band of dentin used in arbitrary calculations of thermal impact. Note its relative size as compared with bur, D—dentin beyond area of calculation, M—area drawn enlarged in Fig. 3B.

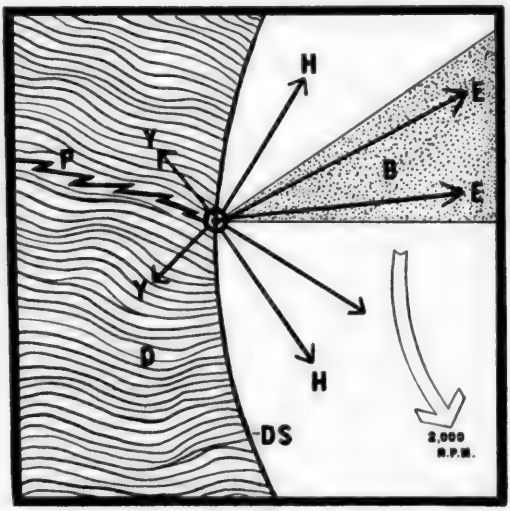

FIG. 4. *Enlarged section from fig. 3.* ⊙—Point of heat impact which travels laterally along dentin surface (DS) at about 25 feet per minute (see text). Note that friction, and therefore, heat and pain, arises at surface of dentin under treatment and thereafter energy is dissipated in all direction. B—cutting blade of steel #558 bur head, E—heat dissipated into steel bur (greatest loss here), H—heat dissipated into debris, moisture, air, during cavity preparation, D—dentin, Y—heat dissipated into dentin (probably least amount lost of 3 available escapes), P—pain sensation on its way through dentin tubules to the central nervous system via the dental pulp.

under identical conditions as above except that only the lower half was used to cut dentin, while the upper half revolved free from frictional contact. Four pounds total pressure with only 3.5 sq. mm. working surface gave a 2.5°C. rise; a caloric output identical with the above experiment when the entire 7.0 sq. mm. cutting surface of the bur was used.

Pressure: Two pounds total operating pressure instead of 4 resulted in an expected rise of only 1.25°C. of the 8 cc. water per minute. This rise remained constant whether the whole (3a) or only half (3b) the bur length was used. In these experiments, however, calculations indicate an increased heat impact per dentin tubule when the cutting or frictional area is decreased. To illustrate:

Exp. 3: 2 lb. pressure —*# 558 steel bur—2,000 r.p.m.*

$$1.25°C. \times 8 \text{ cc. } H_2O = 10 \text{ c per minute} = 0.167 \text{ c per second}$$

Exp. 3a: 2 lbs: 7.0 sq mm. frictional area

$$0.167 \times \frac{1{,}000}{.5 \times 7.0} \div 2.1 \div 0.3 = 75°C. = 135°F.$$

Exp. 3b: 2 lbs: 3.5 sq. mm. frictional area

$$0.167 \times \frac{1{,}000}{.5 \times 3.5} \div 2.1 \div 0.3 = 150°C. = 270°F.$$

Diminishing the total working pressure to 1 pound gave a proportionate drop in calories, but a simultaneous and similar decrease in working bur length maintained an equal heating of each square millimeter of dentin surface.

Bur size: Further experiments with smaller burs all the way down to # 33½ inverted cone and # ½ round burs which have about 0.5 sq. mm. available cutting surface gave limited caloric heat as may be expected, but upon calculating with the working frictional area of such burs, it was disclosed that the *heat impact per dentin tubule* was similar to the results of the *# 558 bur* recordings. With such small burs the caloric generation was only about $\frac{1}{14}$ of that with the # 558 bur, but the heat was impacted upon a proportionately smaller area.

Stones: The work was continued using various carborundum and diamond revolving instruments. The technique of the # 558 recordings was continued. Carborundum and diamond stones gave a thermal pattern similar to steel burs, and since they are generally larger, the total heat was greater, somewhat proportionate to the size or useful frictional surface of the instrument. As with burs, the proportion depended upon the power of the motor, and the tightness and absence of skidding of the engine belt. However, even this moderate increase did not hold for stones over $\frac{5}{8}$" because of drag; their increased circumference induced slippage of the engine belt and slowing of the motor. The greatest heat generation was a rise of 7.8°C. or 14°F. and was caused by a medium size "heatless" stone which measured 2 x 13 mm. Calculating as above: heat generation was 62.4 calories per minute or about 1.0 c per second. The frictional area may

be considered 4 mm. in length, or 2 x 4 mm. = 8 sq. mm., and the depth 0.5 mm., yielding a volume of 4.0 cu. mm.

$$c\ (1.0) \times \frac{1{,}000}{0.5 \times 8} \div 2.1 \div 0.3 = 396°C.\ \text{or}\ 712°F.$$

This figure of 712°F. reflects gentle use of the stone. "Heatless" stone, indeed!

DISCUSSION

Since the heat results from frictional contact of the steel bur with dentin there is possible an alternative hypothetical evaluation. We might consider that the bur gets hot and that the hot blades of this instrument contact the very sensitive dentin fibrils and cause pain. The head of a # 558 bur weighs about 0.02 g. and has a specific gravity (11) of 7.70 compared with water and a specific heat (13) of 0.107. Calculating as:

$$c \times \frac{1{,}000}{\text{wt. steel bur head in mg. (am't. steel heated)}} \div \text{S.G.} \div \text{S.H.} = °C.$$

or

$$.33 \times \frac{1{,}000}{20} \div 7.70 \div .107 = \frac{20.0°C.\ \text{or}}{36°F.}$$

It is probable that both the bur and the dentin are heated and that the surface temperature lies somewhere between the 270°F. rise for dentin and the 36°F. rise for the # 558 bur. It may be argued that the same unit of heat energy, .33c., is being applied to both the dentin and the steel bur and that the unit should be divided. While this may be partly true, the unit was measured by the only method which was found suitable for this experiment, the temperature rise of a stream of water, which incidentally automatically cleaned and lubricated the bur during cutting. A dry or barely moist bur would have generated far more heat. The estimated temperature was also minimized by heat lost from the experimental apparatus itself.

In addition, it is generally recognized that given the same heat source, thin gauge metal gets hotter than heavier gauges, so the thinner blades of the bur get hotter than the body of the head of the bur. Unfortunately, it is the blades which touch the dentin and upon the temper of the blades depends continued cutting efficiency. The generally observed extended life of steel burs when used with the fluid stream is here partly explained. Another factor is the elimination of debris and clogging which mechanically chips and breaks fine steel blades. Also, since the blades get hotter than the head of the bur, so does the actual cut dentin surface of 7 square mm. become hotter than the entire 3.5 cu. mm. All the heat is necessarily inflicted upon the open cut ends of the fibrils. This surface temperature may be many times our estimate. If the bur revolves for more than 1 second another proportionate multiple is secured.

CONCLUSIONS

Thermal changes from steel burs form a regular pattern or coefficient of friction with the caloric heat somewhat proportionate to the frictional surface; the larger

the bur, the more total heat and the greater possibility of pulp trauma, but the same heat impact per dentin tubule from all burs. Although it is generally accepted that the total friction developed by steel burs is proportionate to the working area of the bur head, this is not quite correct because of the intrusion of other factors. As larger burs are used, and the friction increases, slippage of the engine belt and slowing of the motor become evident. Also, the greater cutting surface of larger burs results in distributing the operating pressure over a larger area, and if the total pressure is constant, the total frictional heat is also constant. The total frictional resistance is independent of the area of contact, and for 2 given surfaces such as steel and dentin it depends only on the thrust or force between them (13). The coefficient of friction between dentin and steel is fairly constant, and varies mainly with the type of cutting, i.e.: 1. end cutting or drilling type burs, 2. lateral cutting (most prevalent) which really grind. With a weak dental motor, or a loose belt, it is doubtful whether large burs like the #560 generate more total heat than smaller ones like the #558.

APPLICATION IN OPERATING

Under experimental conditions frictional heat was proportionate to speed and pressure; if 2 pounds pressure was used instead of 4, the heat was approximately halved. While it would seem if speed remained constant and the operative pressure was reduced to 1 pound that the thermal rise would be only 1 fourth of the estimated figures for 4 pounds pressure, conventional operating procedures unfortunately stabilize the temperature at high impact levels. During actual cavity preparation, light bur pressure is usually accompanied by the employment of only a part of the bur length. If engine speed and pressure are constant, the benefits of a decreased working frictional surface are neutralized by the increased pressure per square millimeter; therefore, the use of half the bur length with half the hand pressure will produce an equal heat impact per dentin tubule as when double the hand pressure is used on the entire bur length. The use of half the bur with equal pressure doubles the heat. This partly explains the painfulness of our smallest burs when the heat stimulates nerve tissue to a pain response but may be insufficient to traumatize the pulp, even during abuse.

The above phenomenon is ameliorated in some cases since all pain from cavity preparation is not from thermal impact, but is partly due to mechanical irritation. Smaller burs cause less mechanical traumatization than large ones and as progressively smaller burs are used, a point is often reached where the diminution in pain becomes apparent. Thus, if heat alone were the cause of pain, and speed and pressure were kept constant, the use of the full 3.8 mm. length of the #558 bur would be less painful than the application of only part of its useful cutting surface. Minimal mechanical irritation may fall below the threshold of irritability. However, there are individuals whose threshold of mechanical irritability is so low that they are agonized by the smallest (#½ round or 33½ inverted cone) burs used dry. Most patients of this type affirm the efficacy of "Thermal Control" (14) although some exhibit such low thresholds of irritability that mechanical cutting alone results in maximum pain. When the larger burs (#560, #5, #36, #703, etc.) cause more pain than smaller burs it is because of greater total heat and increased mechanical traumatization.

Dry stones generate more heat than the above recording, and also more than burs of a similar size because of glazing, i.e., the clogging of the stone with debris. Fortunately, most stones are used wet, although it may be suspected that without a constant stream of water, the cutting surface of the dipped and water-glistening stone or disc is usually close to 212°F.

It is interesting to note that with the fluid stream of 8 cc. of water per minute the maximum general temperature rise was 14°F. during normal operating with the larger revolving instruments. This is well within the "temperature tolerance zone", and would not traumatize pulp tissue. Should increased pressure or speed be necessary, a similar increase in the rate of water flow would maintain a constant temperature.

Clinically, 90°–110°F. water is used at a rate of 10–20 cc. per minute. Besides the diminution in pain, increased operative speed, complete pulp safety, and the extended life of burs, the carborundum stones maintain their shape and last a long time. 250°F. oil treatment keeps hand pieces and contra-angles in good condition, and a modified saliva ejector maintains a suitable field of operation. Although operative visibility may be impaired in some cases, it is more often markedly improved because of the darkening of caries when wet and the lack of accumulated debris.

SUMMARY

Heat trauma from revolving instruments may be divided into parts—1. The generation of such frictional heat, of a sufficiently large caloric value, that although distributed over a large area, the sequellae may include pain, dessication of dentin tubules, hyperemia, and possible pulp death. 2. The generation of frictional heat over a relatively small area, of a caloric value sufficient to cause pain by the intensity of thermal impact per dentin tubule, but insufficient to endanger the pulp. The temperature of these small frictional areas exceeds the high tolerance zone limit of 130°F. and ranges from an estimated rise of 67.5°F. to 270°F., depending upon individual operating speed and pressure (1–4 lbs.). These figures are not intended to be accepted literally, but are estimates of potential thermal impact. Because of many variables, and experimental limitations, some of which have been mentioned, it is probable that more accurate figures would be considerably higher.

Since the second type of heat trauma occurs even with the gentlest use of the smallest burs, all cavity preparation by revolving instruments should be performed under a stream of warm water. Rubber dam is contra-indicated except for final observation and minor finishing touches. With "Thermal Control" pain is prevented and the need for anesthesia is appreciably decreased, while operative speed is enhanced and pulp safety from thermal trauma is complete. It should be considered malpractice to anesthetize away pain which need never have been caused.

The specific heat of dentin and of cortical bone may be considered to be 0.3. The work on "Thermal Control" applies not only to the cavity preparation of dentin, but to the use of revolving instruments in bone surgery.

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THE RELATIONSHIP OF PARTICLE SIZE AND OTHER PROPERTIES OF DENTIFRICE INGREDIENTS TO TOOTHBRUSH-ABRASION OF ENAMEL[1]

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We have been engaged for several years in the study of the abrasive properties of dentifrices and their individual ingredients, with the broad aim of establishing the basic factors controlling toothbrush-abrasion. In the first phase of this study, a mechanical brushing device was built through which controlled abrasion of teeth or other materials could be produced. A procedure for using this device on teeth was described in some detail in the first report (1), including factors which must be controlled, such as swelling of teeth, concentration of slurry, variability of teeth, etc. In a second paper (2), the problem of determining the size of particles in powders was considered at some length. From the many such procedures suggested by others, for special purposes, there were selected the microscope visual method, and the Klein surface-increment scale hydrometer technic, for characterizing the powders used in this study. The particle size distributions of a considerable number of powders were measured by these two technics, and the mean and median particle sizes and specific surface areas calculated.

This paper presents results on the correlation of the sizes of the particles in these powders with their abrasive power for teeth. There are also considered the influence of hydrogen ion concentration and of hardness on abrasion. In subsequent papers, the correlation between the abrasion of these powders for teeth and various metal plates and glass will be presented to determine whether the use of these test objects will provide a less variable material for such studies, which can be relied on to predict the abrasion which would be caused by the powders on teeth. As a part of the general program, the abrasive power of some 60 commercial dentifrices has also been reported (3), including some consideration of the newer types of liquid preparations.

W. D. Miller (4) first forcefully attracted attention to the need for studies of this type when he showed that most dentifrices in use in 1907 cut the teeth rather badly when used under conditions not unlike those of their everyday application. He demonstrated that acidic materials enhanced the process of toothbrush "wasting" by extracting the calcium salts from the enamel. He also pointed out, many years in advance of the recent releases on the subject, that anyone brushing

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his teeth daily "will invariably wear away his teeth at the necks inside of a very few years, unless they are protected by healthy gums."

Head recorded, in 1917, the results of brushing teeth with various powders and commercial dentifrices (5). Cementum was abraded much more readily than enamel, but even enamel showed definite wear from some of the more abrasive materials after 10 minutes brushing by hand. He attempted to throw light on the problem of particle size by preparing 3 specimens of calcium carbonate at different rates of precipitation. This gave him a coarse powder with particles ranging from 5.6 to 17 microns and a fine one of 2 to 4 microns diameter. However, these were equally destructive on the enamel, and Head naturally concluded that "it was the chalk itself, and not the preparation, that was responsible for its destructive action." In view of the results to be presented in this paper, it is obvious that his failure to demonstrate differences between the powders resulted from his measuring the teeth to only 0.0001 inch, and from brushing too brief a time to cause an adequate degree of change.

In 1933, Ray and Chaden (6) used a mechanical abrasion machine on which antimony and silver plates were abraded by various specimens of calcium carbonate and calcium phosphate. Since the question of metal plates will be gone into in considerable detail in a subsequent paper, we shall not attempt to discuss here the results of these authors, nor of others who have made similar studies on metals. Wright and Fenske (7) made more extensive quantitative studies on the abrasion of teeth, using the apparatus of Ray and Chaden. As abrasive materials they used calcium carbonate, magnesium carbonate, magnesium oxide, dicalcium phosphate and tricalcium phosphate. The wear of various specimens of these powders on enamel, dentin, and the cemento-enamel junction were compared with that on antimony-blocks. In all cases, the enamel abraded only a small fraction as much as the other two regions. Indications of some of the difficulties in such studies are seen in the fact that, for some of the powders, the dentin was abraded about twice as much as the cemento-enamel junction, whereas with others just the reverse relationship held. These authors stated that they used from 12 to 40 teeth for each measurement, but, since they did not give the standard errors of their values, it is not possible to decide whether these discrepancies are due to variability in the experimental results, or to specific influences of the powders in question. An index summating the abrasion for the 3 structures did not agree well enough with the abrasion of the antimony plates to permit them to conclude that the plates would be reliable test objects for future studies.

M. L. Smith critically discussed their data and conclusions in 1939, and presented the viewpoint that the metal plates were more useful test objects than the former authors had considered them to be (8). He pointed out that hardness of the particles is increasingly important as the hardness approaches that of the enamel. There was also a relationship between the hardness and the size of the particles, since, with the harder materials, small increments in size of the particles caused disproportionate increase in abrasive power. Only in Smith's study was any attempt made to establish accurately the size of the particles in the abrasive

materials, and even here it was not determined what particular measure of their size was the most reliable index of abrasive power.

METHODS

In the present study, teeth were abraded by motor driven brushes running in a slurry of powders in glycerine and water. Seventy-two measurements were the standard for each test, consisting in measuring the wear on the enamel cusps of 18 mandibular molars in a round-robin procedure after 200,000 strokes. The details of this technic have been published previously (1). To minimize the irregularities due to variability of the teeth, a control of calcium carbonate was always included as one of the test powders, and the degree of abrasion calculated as a percentage of this control.

The wet brush alone was shown to abrade to the extent of 57% of the control calcium carbonate. Since, in this report, we are interested mainly in the abrasive power specifically due to the powders themselves, each value presented here has had the effect of the brush subtracted from it, to give the net effect of the powder. The powders used were those described in a previous paper on particle size (2), from which the data on size distributions have been taken for correlation. The abrasion values have standard errors of approximately 15% of the stated values, from which the range of variability has been determined and the degree of reliance of differences between the powders estimated.

CORRELATION BETWEEN ABRASION AND VARIOUS MEASURES OF PARTICLE SIZE

The size of particles in a powder may be expressed in terms of several different measures. The simplest is the median size or diameter of the particle of such size that exactly one-half the particles are larger and one-half smaller than the median. The mean size is obtained by adding the diameters of all the particles together and dividing by the total number to get the arithmetical average. If the powders are evenly distributed, so there is no disproportionate number of any one size, the mean and the median closely approximate each other. The surface area of the powder may also be important in abrasion. This can be expressed either as the average surface area per particle, or more customarily, as the "specific surface," which is the total area per cubic centimeter, or per gram of powder. Cubic measures are those of volume, or weight, and may consist of the median volume per particle, the mean volume, or the diameter of the particle of mean volume. The latter two are simple transformations of each other. Formulas for calculating these various indices are given by Perrott and Kinney (9) and many other authors (2) and need not be repeated here.

In this study we have used the median diameter, mean diameter, diameter of the particle of mean volume and the specific surface for the correlations. All 4 of these were available for the microscopic measures of particle size, where the frequencies of particles of each size are determined. With the hydrometer the quantity measured is the weight distribution, from which the linear quantities cannot be derived, so that only the specific surface and diameter of the particle of mean volume were available for this measurement.

The values secured for these 6 measurements are summarized for 10 specimens of precipitated calcium carbonate in Table I, and for 7 specimens of dicalcium phosphate in Table II. The actual distributions were published in a previous

TABLE I

Correlation between abrasiveness of calcium carbonate on teeth and various measures of particle size

Abrasion values have been reduced by the 57% abrasion produced by brush alone

PRODUCT	ABRASION PER CENT OF CONTROL	MICROSCOPIC				HYDROMETER	
		Median Diam.	Mean diam.	Diam. particle of mean vol.	Specific surface	Diam. particle of mean vol.	Specific surface
		μ	μ	μ	cm.² per gm.	μ	cm.² per gm.
Sturge-70	26	1.7	2.2	4.8	4520	6.7	4870
Snow Top "Regular"	38	1.9	2.8	7.7	2820	3.8	9990
Snow Top "Fine"	43	1.1	1.7	6.8	3180	2.2	19790
Control U.S.P.	43	3.1	3.8	7.3	3020	3.4	14810
Sedimented 1–5	55	2.6	3.0	6.3	3460	4.1	7600
Sturge-50	101	1.7	3.5	15.8	1350	7.8	5970
Snow Top "Heavy"	118	3.8	4.6	8.5	2440	5.7	5640
Sedimented 5–15	246	6.1	13.3	8.9	2450	10.0	2970
Sturge-130	383	7.7	8.2	13.2	1670	11.1	4100
Sedimented 15–30	575	13.1	13.0	17.9	1220	18.3	1570

TABLE II

Correlation between abrasiveness of dicalcium phosphate ($CaHPO_4$) on teeth and various measures of particle size

Abrasion values have been reduced by 57% abrasion produced by brush alone

PRODUCT	ABRAISON PER CENT OF CONTROL $CaCO_3$	MICROSCOPIC				HYDROMETER	
		Median diam.	Mean diam.	Diam. particle of mean vol.	Specific surface	Diam. particle of mean vol.	Specific surface
		μ	μ	μ	cm.² per gm.	μ	cm.² per gm.
Monsanto—"Fines $\frac{1}{8}$ inch"	−2	0.8	1.3	4.7	5300	3.3	20470
Sedimented 1–5 μ	18	1.8	2.8	7.8	3250	5.4	9740
Victor—"Fines $\frac{1}{4}$ inch"	23	3.6	4.1	6.8	3700	6.1	10940
Victor — "Air Blown Dust"	38	1.5	1.8	9.9	3240	1.6	27530
Victor—"Not separated"	50	5.4	7.1	13.7	1830	7.0	7570
Sedimented 5–15 μ	73	5.4	8.0	16.9	1510	12.7	3780
Sedimented 15–30 μ	121	15.6	14.8	22.8	1110	28.5	1820

paper (2). Two samples of dicalcium phosphate of the previous report are not included in Table II. With one, the supply of the powder was not sufficient for the abrasion studies, and with the other a mix-up in the specimens apparently occurred during the studies, making it necessary to discard this material.

In Tables I and II the powders are arranged in order of increasing abrasiveness. With all 6 measures of the particles there is, in general, a tendency for a regular sequence related to that for the abrasion. However, there are many discrepancies in order of size of individual powders and also possibilities of non-linear relationships. Therefore, the abrasion values were first plotted against the size-values on linear, semi-log and log-log coordinate papers to determine visually which method of presentation showed least scatter and closest approximation to linearity. The median and mean diameter values plotted as nearly straight lines on linear coordinates, whereas the volume and specific surface values required log-log plotting to achieve the same end. The 2 measures which showed least scatter of the data were the linear plotting of the median values and

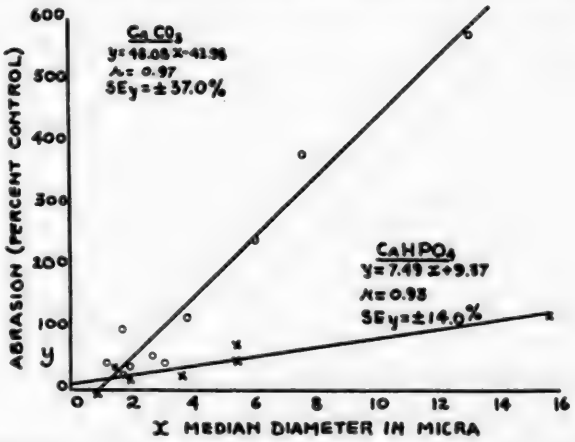

FIG. 1. Relationship of median particle size of calcium carbonate and dicalcium phosphate to abrasion of enamel of human molar teeth. Experimental values are indicated by circles for calcium carbonate and by x for dicalcium phosphate. Lines and statistical constants were calculated from data.

the log-log plots of the diameters of the particles of mean volume, both obtained by the microscope method.

Equations for the relationship between abrasion and median diameter, and log-abrasion and log-diameter of particle of mean volume were, therefore, derived for both the calcium carbonate and dicalcium phosphate powders, using the method of least squares. The standard errors and correlation coefficients were also computed.

For the powders of calcium carbonate, the diameter-mean volume against abrasion formula was: $y = 2.066x + 0.0157$; for dicalcium phosphate, $y = 2.53x - 1.1655$, where x was the log-diameter and y the log-abrasion. The correlation coefficient for these 2 sets of data was very high, being +0.82 and +0.89, respectively. However, the results with the arithmetical plotting of the median particle sizes were still better. In *fig. 1*, the actual values for the 2 materials are plotted and the calculated lines drawn. For the calcium carbonate the for-

mula was: $y = 48.08x - 42.98$; for the dicalcium phosphate, $y = 7.49x + 9.37$, where x is the median diameter in micra and y the abrasion in per cent of the control. Inspection of the figure reveals that the scatter of the data was remarkably low; in fact, the correlation coefficients were +0.97 and +0.93, respectively. The standard error for the abrasion values of the $CaCO_3$ was $\pm$ 37.0% and for $CaHPO_4$ $\pm$ 14.0%, indicating that the data could be expected to deviate from the calculated abrasion within these limits, according to the usual probability distributions.

Probably the most accurate estimate of the relative abrasiveness of calcium carbonate to dicalcium phosphate is given by the ratio of the x coefficients in the equations; that is, the abrasiveness is as 48.08 to 7.49 or 6.4 to 1. Such a ratio is the only generally applicable ratio of comparison, since comparing the degree of abrasion at any given particle size will give ratios which will be different for every different diameter of particle selected.

ABRASION BY SPECIAL DENTIFRICE INGREDIENTS

There are many ingredients in dentifrices in addition to the calcium carbonate and dicalcium phosphate discussed above. It was not feasible to measure enough different samples of each one of these to construct curves of abrasion, and, therefore, their abrasive power, as determined by the slopes of the curves, was not established. However, individual specimens of 15 compounds were measured, and the results are summarized in Table III. It appeared to be a useless procedure to measure the particle sizes of the more soluble compounds, since their crystals would partially dissolve or disintegrate in the liquid used to make the slurry, and the particle size measurements would be meaningless. However, seven of the relatively insoluble powders were measured for size as shown in Table III.

By comparing the amount of abrasion of a given powder with that caused by calcium carbonate of the same particle size, a general idea can be obtained as to whether it is more or less abrasive than the calcium. Probably the data may be stretched to yield more information if it be assumed that the curves would pass through, or at least very close to, the point of origin of the graph, as do the data on the other 2 powders in *fig. 1*. On this assumption, the slope of the hypothetical curves would give an idea of the approximate range of abrasiveness relative to the 2 more thoroughly studied powders. With magnesium carbonate, for example, an abrasion of 27%, at a particle size of 9.8 micra, was much less than that of the other two. Tricalcium phosphate was very close to dicalcium phosphate, as was also fuller's earth. Highly abrasive were flour of pumice and silica, as might be expected from common experience. From the entire list of compounds it appears highly probable that only the last 2 were more abrasive than the chalk when the probable particle sizes are considered.

HYDROGEN ION CONCENTRATION

Nearly all dentifrice ingredients are alkali buffers, since it is generally realized that teeth are readily damaged by acid. However, Hansen and Fosdick (10)

have demonstrated that considerable degrees of acidity may be present in some dentifrices.

In order to determine the actual acidity under our conditions, the pH of the slurry was measured on several occasions with a Coleman glass electrode. Since saturated solutions of the powders were being used in a liquid consisting of equal volumes of glycerine and water, the usual dissociation constants could not be

TABLE III

Correlation between abrasiveness of various dentifrice constituents on teeth and their physical properties

Abrasion values have been reduced by 57% abrasion produced by brush alone

COMPOUND	FORMULA	ABRASION IN % OF CONTROL	pH OF SLURRY	MOH HARDNESS	DIAMETER OF PARTICLE	
					Median microscope	Mean volume hydrometer
					μ	μ
Magnesium carbonate U.S.P.	$MgCO_3$ with $Mg(Oh)_2 \cdot 5H_2O$	27	9.7	2.5	9.8	2.2
Magnesium oxide heavy, U.S.P.	MgO	29	10.0	2	8.0	136.0
Tricalcium phosphate, U.S.P.	$Ca_3(PO_4)_2$	34	6.6	4-5	5.6	8.4
Magnesium oxide light, U.S.P.	MgO	41	10.3	2	5.2	3.3
Precipitated chalk, U.S.P. control specimen	$CaCO_3$	43	8.5	3	3.1	3.4
Sodium phosphate monobasic, U.S.P.	NaH_2PO_4	43	3.6	1		
Sodium phosphate dibasic, U.S.P.	Na_2HPO_4	43	8.7	1		
Dicalcium phosphate	$CaHPO_4 \cdot 2H_2O$	50	6.4	3	5.4	7.0
Sodium chloride, U.S.P.	$NaCl$	62	5.6	2.5		
Fuller's earth		65	7.8	2.5	8.8	22.5
Magnesium trisilicate, U.S.P.	$2MgO \cdot 3SiO_2 \cdot nH_2O$	75	8.2	3		
Sodium metaphosphate	$(NaPO_3)_n$	104	3.2	2		
Sodium borate, U.S.P.	$Na_2B_4O_7 \cdot 10H_2O$	105	9.1	2.5		
Sodium perborate, U.S.P.	$NaBO_3 \cdot 4H_2O$	105	7.2	3		
Pumice flour	Aluminum potassium & sodium silicates	4325	8.1	6		
Silica "240 mesh"	SiO_2	5688	7.8	7	10.5	21.1

used to predict the acidity. The electrode assembly was tested for proper functioning at the beginning and end of every series of readings by measuring a known standard buffer solution.

The glycerine-water mixture had a pH of 6.35. This slight acidity, due to dissolved impurities in the glycerine or water, represented such a slight buffer power that it was considered negligible in the presence of the buffer materials.

The most acidic compound was sodium metaphosphate. This salt produced a relatively large amount of abrasion, although, in the absence of particle size measurements, it is uncertain whether it was more harmful to the teeth than other powders of the same particle size. Gordon (11) reported that the tetrametaphosphate forms complex soluble ions with the calcium of the teeth, and many industrial uses of the hexametaphosphate depend on its power to bring calcium and magnesium salts into solution. The influence of this compound needs to be more extensively investigated, since there is the possibility that a surface softening might promote polishing of the enamel without leading to objectionable loss of tooth substance.

The other acid material was sodium acid phosphate, with a pH of 3.6. This had an abrasive power equal to that of the control calcium carbonate. Since these crystals are very soft and friable, it is probable that the acidity played a role in causing the degree of abrasion observed. Sodium perborate is ordinarily considered to be a definitely alkaline compound, which may even be caustic because of the hydroxide it liberates. There was observed here a nearly neutral pH, indicating a decided shift toward the acid side from the expected value. This was presumably due to oxidation of the glycerine to organic acids with a resultant partial neutralization of the alkali. Both the perborate and sodium borate appear to be more abrasive than would be expected.

HARDNESS OF MATERIALS

Tests for the hardness of the ingredients were rather imperfectly carried out, using the Moh scale. The numerous methods for measuring hardness available for metals were not applicable to the fine powders studied. Except for pumice and silica, which are much harder than all the others used, there appears to be no clear correlation between our data on hardness and the degree of abrasion. We believe this reflects on the accuracy of the Moh number determinations made by us, or on this method of measurement, rather than refuting the existence of a relationship between hardness and abrasion. It is, however, obvious that such a relationship cannot be satisfactorily established until special attention is given to comparing only powders of identical particle size distributions, on the alkaline side of neutrality, with hardness values which have quantitative significance rather than merely index numbers like the Moh scale.

McGehee (12) states that the hardness of enamel on the Moh scale is 5.5 to 7, and of cementum and dentin, 3.5–5. Only pumice and silica were found by us to be harder than enamel, and these 2 compounds cut the teeth markedly. Possibly, if the powder is softer than the tooth structure being abraded, the exact degree of hardness of the powder would become a relatively unimportant factor considering all the elements which might be active.

That more work on this phase of the problem needs to be done is indicated by the unsatisfactory state of engineering knowledge on this subject. Thus, Marks, in a standard reference book (13) states: "ability to resist abrasion involves toughness as well as hardness and cohesion, and it is not necessarily indicated by a hardness number." Batson and Hyde (14) made the statement that, while

abrasion from wear goes up in inverse proportion to the Brinell hardness, this cannot be used to predict abrasion because of many materials which do not fall into the series.

DISCUSSION

It is believed that the results of this study have shed some light on the general problem of what type of measurement of particle size will be useful in predicting abrasive power. It was demonstrated that all the various methods can give some information, but that the median size measurement, which is arrived at most simply by the microscopic technic, has the highest reliability. The measurement of the size distribution and abrasion of 2, or better 3, specimens of a given chemical agent will permit drawing a curve of abrasion against size of particle. The slope of this curve appears to be the most reliable index of abrasive power, and can be used for comparison of different powders of varying particle size.

However, the complexity of factors affecting the degree of abrasion are such that much more rigid control of all variables must be instituted than has been commonly done previously in dental researches. Even when all the apparent variables are standardized, there is no certainty of being able to confidently predict the outcome of the measurements. That research on teeth is not unique in this respect is indicated by the following quotation from Morse's "Textbook of the Materials of Engineering" (15): "In considering the effect of hardness on wear it must be kept in mind that hardness is a term used to cover several quite different properties of metal. Wear tests must, in general, be accelerated tests, and a wear test of two metals under a heavy load is not at all certain to rate them in the same order of merit as a longer test under a lighter load. Then too, wear tests of materials against abrasive surfaces may give quite different orders of merit. In tests on gears there seemed to be a sort of "wear limit"—a critical intensity of load below which wear was very slight indeed. The relative values of "wear limit" for various metals could not be predicted from their behavior under a short-time heavy load test. The development of reliable wear tests is one of the unsolved problems in the study of engineering materials."

CONCLUSIONS

1. The abrasion of the enamel of human teeth and of 10 samples of calcium carbonate and 7 samples of dicalcium phosphate of known particle size distributions has been measured under carefully controlled conditions.

2. Measurement of the median particle size of the powders is highly reliable in predicting abrasion.

3. The best measure for comparing the abrasive power of different chemical materials is to compare the slope of the curves relating their median particle sizes to determined abrasion. Comparison of the relative abrasiveness of any two such powders, even at the same particle size, will give ratios which will not hold for any other size particle, unless the curves happen to be parallel.

4. According to this criterion, dicalcium phosphate is about one-sixth as abrasive as precipitated calcium carbonate.

5. Limited data are presented relaying the abrasiveness of fourteen other materials to their particle sizes, to hydrogen ion concentration, and to Moh hardness numbers.

6. The data indicate, but do not prove conclusively, that the Moh hardness number gives little information as to prospective abrasion of enamel for substances softer than enamel. However, pumice and silica, with Moh numbers higher than enamel, were also highly abrasive.

7. There are many unsolved aspects of abrasion deserving extensive study, among which are the influence of pressure, the type of bristle in the brush, differences produced by lubrication, the critical level of hydrogen ion concentration, the optimum degree of abrasion for satisfactory dental hygiene. Still unanswered is the very important problem whether the desirability of a dentifrice should be judged by its effects on enamel or those on dentin or cementum. The increased interest in this field of research indicates that some of the answers should become available in the not too far distant future.

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INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the New York Section[1]

June 8, 1943[2]

Compiled by FRANCES KRASNOW, PH.D., *Editor, New York Section*
Guggenheim Dental Clinic, New York, N. Y.

1. PULP DIAGNOSIS. *Alexander Seelig, D.D.S.,* Montefiore Hospital and School of Dental and Oral Surgery, Columbia University, New York.* An attempt is made to understand the histology and the pathology of the dental pulp by studying first the general condition and the treatment accorded the patient and then secondly the specific environment of the particular tooth. This was done by making up a chart upon which was recorded the chief complaint, the history with all the tests that are commonly made in a dental practice, physical examination of the tooth and gingiva, clinical diagnosis, x-ray diagnosis, treatment, and extraction findings. Then the medical chart of the patient was combed for all related conditions that may have had an affect upon the pulp. Particular attention was given the blood pressure, blood counts, heart, lungs, kidneys, diseases and basal metabolism. The extracted teeth were then sectioned in toto, without cracking or cutting the tooth, so as to avoid the possibility of losing any relationships. The sections were then studied and an attempt was made to correlate the pathosis that was found and the clinical dental diagnosis. This turned out to be a difficult and unproductive endeavor. The reason that this attempt was a failure became apparent when the cases were grouped not according to the dental lesion but according to the medical diagnosis and the medical treatment accorded the patient. For then the tooth was recognized as a specialized organ first affected by the changes that went on through the organism, due to the general condition of the patient, and the treatment that he had received; and secondly, affected by the special conditions and diseases that the tooth was subject to.

2. TUBERCULOUS PERIAPICAL GRANULOMATA. *Ralph Howard Brodsky, D.M.D., and Jack S. Klatell, D.D.S.,* Division Oral Surgery, Sea View Hospital, Staten Island, New York.* At this hospital, one of the world's large tuberculosis hospitals, we conducted a study of periapical granulomata taken at random. The patients included were adult males and females manifesting varying degrees of generalized tuberculous processes. Pathological sections of 362 of the granulomata revealed the presence of tuberculosis in 8% of the cases. The mode of

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[2] Presiding—John Oppie McCall, Guggenheim Dental Clinic.

[*] Guest.

entry of the tubercle bacilli in most cases was probably through caries to the pulp and then periapically. Some cases were infected subgingivally, while others may have become involved by means of a hematogenous spread. A point of importance concerning this condition, aside from the high resistance it would offer to root canal therapy, is its danger, if undiagnosed, as a residual of focal infection which can flare up in what might be an otherwise arrested or cured case of generalized tuberculosis. It may possibly be found in our office patients who are not aware of having any tuberculous process.

3. THE EPITHELIAL SHEATH: HISTOGENESIS AND FUNCTION. PART 2. *Moses Diamond, D.D.S., and Edmund Applebaum, D.D.S., School of Dental and Oral Surgery, Columbia University, New York.* The complete cycle of the histogenesis and function of the epithelial sheath, the formative organ of the root cannot be demonstrated in a single phase of development of the tooth. It has to be observed, instead, in a number of different phases or stages of development. Observing the phenomenon at isolated stages accounts for the widely divergent conclusions in the literature as for example, those of von Brunn and Mummery. The true histogenesis can only be observed at what we characterize as the first stage, even before the matrix stage of enamel formation is completed. This stage was presented in a previous publication. The second stage, now being presented, shows the classic picture of Mummery, who believed that the inner enamel epithelium played no part in the histogenesis of the epithelial sheath, but only the outer enamel epithelium. This second stage gives that illusion for one may observe in this stage that the ameloblasts or inner enamel epithelial cells, terminate at the cervical line where the enamel matrix terminates, and appear to have no organic connection with the epithelial sheath. The organic connection between them is now disrupted and in a state of atrophic degeneration.

4. THE RAMUS AS A FACTOR IN THE DEVELOPMENT OF THE DENTAL HEIGHT. *Moses Diamond, D.D.S., School of Dental and Oral Surgery, Columbia University, New York.* It is generally believed that the force of clinical eruption of the teeth is a primary and active factor in increasing the vertical dimension of the dental height. The force of clinical eruption, it is maintained, "wedges" the jaws apart, steadily increasing the intermaxillary space and thereby permitting vertical growth of the body of the mandible and of the maxilla. Schour and Massler support this view with deductions based upon experimental growth studies. "Normally, the vertical height of the jaws and the length of the arch are increased by the eruption of the teeth. In hypopituitarism the failure in eruption results in an insufficient increase in vertical height and arch length so that the teeth, the crowns of which are normal size, may become crowded or submerged." Thus, in hypopituitarism, the retardation of growth in vertical dimension of the dental height is attributed to the force of eruption as the primary effect resulting in a submergence of teeth and only secondarily resulting in a retardation of vertical-length-growth. Evidence is presented both in normal physiologic growth processes and in disturbed or retarded growth processes that the primary factor in increasing the vertical dimension of the dental

height is the growth in the length of the ramus. Growth takes place in alternate processes of acceleration and retardation. The accelerated spurts in ramus-length-growth just precede the vertical growth of the body of the maxilla and of the mandible and the clinical eruption of the teeth. Increase in length of the ramus lowers the body of the mandible, increasing the intermaxillary space and permitting vertical growth of the body of the maxilla and of the mandible and the clinical eruption of the teeth. In the first 5 years of life this mechanism is additionally aided by the downward growth of the temporal bone into which the condyle head of the ramus is articulated. In this way the entire mandible is additionally lowered during the first years of life. Delayed accellerated length-growth-spurts show a contact relation between the edentulous posterior gum pads or an absence of an intermaxillary space. The growth and development of the teeth is normal for the chronologic age and although every other indication exists for imminent eruption of those teeth, they are unable to erupt because of an absence of intermaxillary space due to a prolonged retardation of an accellerated growth spurt in ramus length. The roots of these teeth, too, grow deeper into the jaw and the epithelial sheath ceases to be a relatively fixed point. It is concluded that the force of eruption plays no part in increasing the dental height. The growth syndrome in hypopituitarism effectively supports these findings, for it is well known that pituitary deficiency primarily affects skeletal growth and therefore also retards the ramus-length growth. The developmental processes of the teeth remain comparatively unaffected. Only secondarily is the clinical eruption of the teeth interfered with. The length-growth of the ramus is therefore regarded as the "pace-maker" in the development of the dental height. Additional evidence is presented that retardation in ramus-length-growth is an important etiologic factor of a large group of resultant malocclusions. The anterior teeth continue in eruption despite retardation of ramus-length-growth. This produces an anterior overbite relation which interferes with the forward growth of the anterior region of the mandible.